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BULLETIN

OF

THE NEW YORK ACADEMY OF MEDICINE

MARCH, 1929

INSTITUTED 1847

PUBLISHED MONTHLY BY
THE NEW YORK ACADEMY OF MEDICINE
Fifth Avenue and 103rd Street
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BULLETIN OF
THE NEW YORK
ACADEMY OF MEDICINE

VOL. V. MARCH, 1929 No. 3

THE SKIN AS A SHOCK TISSUE[1]

ARTHUR F. COCA

(Delivered before The New York Academy of Medicine, December 6, 1928.[2])

I have had to pay my respects to this word allergy on one or two occasions and I think I ought to explain that I did not myself select the term for the present program. As you know, Von Pirquet invented the term from the Greek words meaning altered re-activity and he intended it to cover increased re-activity and not lessened. Doerr took it up, applied strict etymology to it, and included under it conditions of lessened re-activity. Thus he placed with hypersensitiveness such conditions as immunity to diphtheria on the ground that it was specific and the tolerance to morphine of the morphine addict, which then made something of a little waste basket of the term; certainly it lost its real usefulness by this etymological extension. In this country the term of course does not include these two added conditions of lessened re-activity. No one thinks of immunity to diphtheria or the non-specific tolerance for morphine as allergic. In fact, some in this country would confine the use of the term to the human forms of hypersensitiveness while others would include also anaphylaxis. In view of this difference in the use of the term, Karsner and Ecker have said they preferred not to use it at all and I really cannot see how it can be scientifically used. The practical medical man,

[1] From the Department of Bacteriology and Immunology, Division of Immunology, in Cornell University Medical College, New York City, and the New York Hospital, New York City.

[2] Program presented in coöperation with the Section of Dermatology and Syphilis.

however, does use it and will continue to use it as the older physicians use the expression "torpid liver," without wishing to know what that might mean and certainly not caring.

In introducing the subject stated in the program, I should like to talk for some time on the subject of specific hypersensitiveness. Medical progress can not be made without classification. I might recall that we did not know anything about hay fever until we separated it from the common cold, and now more recently we have found that we could not learn much about hay fever until we separated it from anaphylaxis. And so, as a matter of fact, we have found the way to a clearer understanding of hypersensitiveness by distinguishing the five different forms that are shown on the chart. *Anaphylaxis* is an experimental condition of hypersensitiveness in lower animals, so far not found in human beings. The *hypersensitiveness of infection* of the tuberculin type, with which you are all familiar, occurs in animals or in man infected with tubercle bacilli or infected with *Bacillus mallei* and with spirochaetae and with other infectious organisms, which are particularly interesting to the dermatologist.

Atopy is a term covering the several inherited conditions of human sensitiveness. Those that are now well recognized in this class are asthma and hay fever and I think that from this evening's analysis we will be able to add to that with some certainty specific eczema. By specific eczema I mean eczema due to hypersensitiveness to some particular substance, usually a food substance and brought in contact with the skin after ingestion.

Dermatitis venenata is a condition well known to the dermatologist, affecting 60 per cent of human beings over five years of age that have been brought in sufficient contact with the excitant. In this country the most common excitant is poison ivy. Children under five years of age are not so much affected and certainly under three years they are only very rarely affected. *Serum disease* and *drug idiosyncrasy*, also acquired conditions, for the pres-

ent are included under the category of specific hypersensitiveness, neither of them probably due to antibodies.

I should like to speak about the hereditary factor in atopy; *i.e.,* on the question of how heredity governs the occurrence of asthma and hay fever and allied conditions. In the first place, I ought to remind you that these facts have been discovered largely through the researches of Dr. Robert A. Cooke and his co-workers, Dr. Vander Veer and Dr. Spain, and have been confirmed by Adkinson and more recently in a study of 1,000 cases by Ray M. Balyeat, who confirms the original findings of Cooke and his colleagues in every respect and has added an important point to be mentioned later. I should say also that the data as well as the conclusions of the studies of Cooke were submitted before publication to well known geneticists including Morgan, Davenport and Conklin, all of whom accepted the conclusions and considered it one of the best studies of the inheritance of pathological conditions in human beings.

The first way in which the influence of heredity is seen in hay fever and asthma is that where both antecedent lines are affected about 70 per cent of the children will be affected; this must be taken in contrast with the 7 per cent of the general population that are affected by these conditions. If the factor here is a Mendelian dominant we should expect 75 per cent of the offspring to show the condition. Secondly, if both lines are affected, the children are more likely to begin to show symptoms in early life (72 per cent by the age of 10 years) and all of those that are to be affected will show symptoms by the 35th year. Where only one antecedent line is affected the first symptoms may appear as late as at the age of 70. Thus we see that inheritance controls the age of onset of the condition, and since some of the individuals become sensitive to only one substance with which they come in daily contact it is necessary to conclude that the substance to which they are going to be sensitive is determined also by inheritance. The third way in which heredity influences

the occurrence of asthma and hay fever is that if the child is born in a family in which there is a pure hay fever lineage he is much more likely to be affected with hay fever than with asthma, and vice versa—a child of a pure asthma lineage is more likely to be affected with asthma than with hay fever. Finally Balyeat has found that a child subject to a bilateral hereditary influence will more likely show multiple sensitiveness; *i.e.,* to several substances, than one subject to a lesser degree of hereditary influence.

If bronchial hay fever and asthma are subject so completely to heredity, what role does contact play in the establishment of this category of hypersensitiveness? It used to be thought that a person became sensitive to food because he had some anomaly of the gastro-intestinal tract allowing the passing of undigested protein through the mucous membrane into the blood where it acted as an antigen and sensitized the individual as an injected protein does the guinea-pig. Dr. Matthew Walzer, by a very ingenious method, has shown that in at least 90 per cent of people the normal gastro-intestinal tract is permeable to unaltered protein of various kinds, such as egg white, nut, fish, etc. Previous to that, Anderson and Schloss had shown that milk protein of the cow and egg white passed unaltered through the normal gastro-intestinal tract of children, because when they fed those two materials to babies they could often find antibodies (precipitins and anaphylactic antibodies) in the blood of those babies. Therefore throughout life unaltered foreign proteins are normally passing through the gastro-intestinal tract. Yet hypersensitiveness to these food proteins occurs only in those that are of an atopic lineage. Furthermore, asthma and hay fever are both often due, perhaps most often, to substances with which one comes in contact by inhalation. It was once thought that sensitization to these substances was due to an abrasion or some disease of the skin. It was noticed (and this may have been a basis for the belief) that children having eczema were likely later to develop asthma or hay fever. However, we see now that contact

plays only a secondary role in the etiology of these conditions. In the same sense we have to interpret the studies of Ratner and his associates on the passing of antigens through the placenta into the child from the mother. It would not seem to make any difference whether the antigen reached the child through the placenta of the mother or through its own gastro-intestinal tract or through its own respiratory tract. The route of its entrance into the individual certainly can have no special importance in the establishment of sensitization and once you have discovered the primary exclusive importance of inheritance in the establishment of hypersensitiveness, then any other consideration must take secondary place.

However, contact is probably necessary to the development of hypersensitiveness in a person that is born of atopic lineage. There are many people living in Europe who never come in contact with ragweed (which does not grow there but grows here), and who, if they came to this country, would become sensitive to the ragweed pollen. We know from studies here that about one-fourth of all subjects of hay fever sensitive to timothy pollen will be sensitive also to ragweed pollen. There are many cases of hay fever individuals sensitive to timothy pollen in Europe; one-fourth of these are potentially ragweed sensitive. Until they come in contact with ragweed pollen, the sensitiveness cannot develop. I would like to emphasize that not all forms of human hypersensitiveness are subject to inheritance; one can see this best by reference to the hypersensitiveness of the tuberculin type. Most individuals who become infected with the tubercle bacillus will develop this form of hypersensitiveness. Also dermatitis venenata, affecting 60 per cent of the population, cannot exclusively affect people of atopic lineage. In fact, we have tested a number of hay fever and asthma subjects with ivy extract and have found just as many of them insensitive to the extract as are found among non-atopic persons.

Having called attention to the importance of heredity in the establishment of atopy, we can now take up the

analysis of specific hypersensitiveness in general and especially compare anaphylaxis, which is experimental in animals, and atopy which is inherited in human beings.

There are two keys necessary to the understanding of specific hypersensitiveness. The first is the idea of the specific mechanism and the second is the idea of the shock tissue. In anaphylaxis the specific mechanism is the specific antibody, *i.e.,* anaphylactic antibody in the animal. These antibodies are produced actively by injecting the animal with a foreign protein, and their presence in the blood can be shown by injecting the serum into a normal animal, which almost at once becomes sensitive to the same substance that the original animal was sensitized to. This then is the specific mechanism in anaphylaxis; it is produced in lower animals and also in human beings. When produced in human beings it is not accompanied by clinical hypersensitiveness in the individual. This was first reported by Schloss. The shock tissue in the lower animals in anaphylaxis is unstriped muscle; anaphylactic antibodies from any animal, whether human being, dog, bird, or rabbit, are capable of sensitizing passively the unstriped muscle of the guinea-pig. The classical test object in the guinea-pig for the identification of the anaphylactic antibody is the uterine strip which is almost pure unstriped muscle.

In atopic hypersensitiveness, *i.e.,* asthma and hay fever, the specific mechanism is not the anaphylactic antibody; it is another sensitizing body, which differs characteristically in almost every respect from anaphylactic antibody, and which we called the *atopic* reagin. The atopic reagin can be demonstrated in every case of bronchial asthma and hay fever that is subject to an identified antigen. It is demonstrated in the blood of these individuals by injecting the serum of the sensitive person into the skin of a normal person and testing that site with the known excitant. In every case if proper conditions are observed a wheal will result from the test injection in the sensitized site. The reagins differ from anaphylactic anti-

bodies, first in the fact that they are not capable of sensitizing unstriped muscle. Since anaphylactic antibodies do not sensitize the human skin, the two sensitizing substances are in this respect diametrically different. They differ also in the fact that the reagins will not neutralize the respective antigen. Every other know antibody neutralizes and eliminates its respective antigen. Antitoxin neutralizes toxin and stops its toxic effects; precipitin precipitates its antigen and anaphylactic antibody neutralizes its antigen; but reagin has no effect whatever on the corresponding excitant. This fact explains why, although an individual may have many reagins in his blood, the injection of a small quantity of the excitant under the skin will be carried through the blood to the sensitive tissues and there produce the constitutional reaction. If there were anything like that amount of ordinary antibody in the blood of that individual, the antigen would never reach the susceptible tissues. Let us take antitoxin, for example: Only 1/100 of a unit of antitoxin per cm. is needed in the blood of a human being to render that person insensitive to diphtheria. Toxin injected into the skin of such a person will be neutralized in the blood before reaching the susceptible tissue. The excitant in hay fever and asthma on the contrary is not affected in the slightest degree by any quantity of reagins with which it comes in contact in the blood on the way to the susceptible tissue.

The atopic shock tissues in the human being differ from the anaphylactic shock tissue (unstriped muscle) in two respects. In the first place, the atopic shock tissues are not susceptbile to specific influences in the normal, that is, the non-atopic person; secondly, not all of the several shock tissues (the ophthalmic, bronchial, nasal, gastro-intestinal and cutaneous) are susceptible in every atopic individual; one may have asthma only or hay fever only, and this, as we have already mentioned, is subject strictly to inheritance.

We can now turn to the stated subject, which is the skin as a shock tissue. It is a remarkable fact that the

skin exhibits itself as a shock tissue under varying circumstances and with very different reactions. Let us consider, for example, the wheal. The wheal can occur under four different circumstances, first, by simply sensitizing a site in a normal skin and testing it with the excitant to which the individual from which the serum was taken is sensitive. We sensitize with serum from subjects of hay fever, asthma or eczema. We then inject the excitant into the skin in that site and a wheal is produced. Secondly, the atopic individual himself, whether he suffer from asthma, hay fever or eczema, if tested intradermally with the excitant of his particular form of hypersensitiveness will exhibit a wheal at that site. This wheal is produced because the skin is constantly sensitized with the individual's reagins circulating in the blood. We do not have to assume any specific hereditary quality of the skin in the production of a wheal under these circumstances. Thirdly, an individual subject to hay fever or asthma after subcutaneous injection of an overdose of the excitant will sometimes develop a generalized urticaria. Here again for the same reason as mentioned above, it is not necessary to assume a special inherited quality of the skin in the production of urticaria. Finally, if the patient is suffering from asthma due to some food, when that individual ingests the food an urticaria may take place; again, not on account of any inherited quality of the skin but because the skin is sensitized by circulating reagins in the blood.

In *dermatitis venenata* we have an exhibition of the skin as a shock tissue with a lesion that is characteristically different from the wheal. There is no difficulty in distinguishing this lesion from that of the wheal. What the difference is due to here, I am not prepared to say, but certainly it need not be considered as due to any inherited quality of the skin, because *dermatitis venenata* occurs in non-atopic individuals as well as atopic. Furthermore, the *tuberculin reaction* in the skin is different from either the wheal or the lesion of *dermatitis venenata.* The cause of this difference has not been really studied, except-

ing so far as to observe that histologically the tissue of the tuberculin reaction resembles to some extent tubercular tissue.

Now we come to the question whether in the skin there exists a form of hypersensitiveness that can be distinguished from the three just described and can be considered as due to some inherited quality of the skin, and the only form that meets these requirements is specific eczema. In specific eczema we have a lesion that is characteristically different from the wheal. There may be gradations between the two, but an outspoken eczema can always be distinguished from urticaria. Comparing these two lesions of hypersensitiveness, we see that they are both due to the same kind of excitant; indeed, eczema and urticaria may be produced in two individuals by the very same food. Secondly, the reagins in the two instances are the same; they are not distinguishable; *i.e.,* reagins in eczema if transferred to normal skin sensitize the skin but when you test that site, you do not produce eczema, you produce a wheal. Hence, in every respect in which they can be studied, the reagins causing specific urticaria are identical with those causing specific eczema. This leaves us only one possible explanation of the difference between the two lesions, since the causes are otherwise the same, and that must be a hereditary peculiarity of the skin in the eczematous individual. When we examine the eczematous individuals in this respect, we find considerable suggestive evidence of a hereditary influence. In the first place, the eczema of this kind occurs often in children that have either asthma or hay fever or who, after they have outgrown the eczema, develop asthma or hay fever. Furthermore, there is very often a positive atopic family history in these cases.

The lesson then in this comparison is that in *specific eczema we have a distinct pathological clinical entity which is separable from the other forms of hypersensitiveness of the skin.*

ALLERGY IN SKIN DISEASES

SIGMUND POLLITZER

(Delivered before The New York Academy of Medicine, December 6, 1928.*)

It was no doubt noticed even by primitive man that the ingestion of certain articles of food was followed by abnormal or unusual effects in some individuals, abnormal or unusual inasmuch as these articles of foods did not produce similar effects in most people. It is a matter of common experience that shell-fish, strawberries, etc., while useful and agreeable articles of food in general, when introduced into the alimentary canal in a small number of individuals are regularly followed by distressing cutaneous symptoms. Most people can spend a day in our fields or forests without any untoward effects, but a few will suffer within a few hours from an inflammation of the skin, resulting from contact with certain plants. Most druggists can handle novocain, mercury, ipecacuanha, etc., with impunity but a small number will develop a dermatitis from contact with one or other of these drugs. Masons may handle cement, bakers flour, all their lives, but in a few instances, an insignificant percentage of people engaged in these occupations, a dermatitis of the hands and face results from contact with these substances, a dermatitis which recurs on each renewed contact with them.

These are simple and well-known examples of a condition, which until twenty years ago, was explained as due to idiosyncrasy. I say explained, but of course nothing was explained by using a word which implies merely that a given effect was unusual and peculiar to an individual. On the other hand, the use of the term allergy or atopy for these unusual effects implies a definite theory of their pathogenicity, takes them out of the order of unexplainable mysteries, and groups them with a class of phenomena which may be the subject of exact laboratory experiment.

Under allergy I understand a state of hypersensitiveness which results from the parenteral introduction—

* Program presented in cooperation with the Section of Dermatology and Syphilis.

through the skin or through the mucosae—of substances foreign to the organism, bringing about changes in certain cells, so that thereafter, the organism responds to renewed introduction of these substances in a peculiar and specific manner. I pass over the question whether this hypersensitiveness is always the result of an immune-biological process like that which follows the injection of a foreign protein into the circulation or not. The special workers in this field are not agreed on this question. I am using the term allergy in this paper without prejudice.

Certain it is that an enormous number of substances of the greatest variety, ranging in chemical constitution from the most complex organic molecules to the most simple inorganic bodies, are capable of inducing a state of specific hypersensitiveness.

The conception of allergy has profoundly altered our views of the pathogenic mechanism of many diseases. Its most brilliant results have been achieved in the field of two common diseases affecting the mucosa, hay fever and asthma, both of them conditions in which certain diseases of the skin are frequently present. The frequent association of urticaria, prurigo and eczema with asthma and hay fever has long attracted the attention of clinicians, but in the absence of a clue to a causal connection, it was regarded as merely a coincidence or dismissed as an inexplicable phenomenon. It has been shown that hypersensitive individuals are commonly hypersensitive to more than one substance. It is not uncommon to find that an allergic individual responds to cutaneous tests with a pronounced positive reaction to a great variety of substances. In a case of hay fever with sensitization dermatitis dating from infancy, Dr. U. J. Wile (personal communication) found that ninety out of a hundred of the various articles tested gave a positive reaction. Matthew Walzer has recently shown in an ingenious and carefully planned study that, contrary to the commonly accepted belief of physiologists, whole or but partly altered molecules are almost constantly passing through the alimentary mucosa into the

blood in some individuals. Such facts seem to indicate that the mucosae of the respiratory tract and of the alimentary canal in some individuals are peculiarly permeable to foreign substances, so that hypersensitiveness may be established to an enormous number of common articles. It has been definitely established that foreign proteins readily pass through the milk glands and through the placenta so that sensitization may occur in earliest infancy or even in utero.

Studies in the heredity of skin diseases in man present peculiar difficulties. Skin diseases as a rule are not of such importance as to make a marked impression on the memories of the patients' descendants. Then, too, they are often regarded as something to be ashamed of. They are not talked about. It is but rarely that we find an example of family hypersensitiveness such as Bloch reports, a mother, two daughters and two grand-children, hypersensitive to primrose. With asthma the condition is quite different. The great suffering, the profound disturbance occasioned by an attack of asthma in an individual, cannot be hidden from the family. We are therefore able to get a history of asthma often extending through several generations. The heredity of asthma following Mendelian laws has been definitely established in an enormous series of cases, beginning with the work of Coca and Vander Veer. In view of the many close relationships between asthma and some skin diseases, it would seem likely that cutaneous hypersensitiveness too is an hereditary character.

The diseases of the skin which we have come to regard as due to hypersensitiveness are urticaria, angioneurotic edema, prurigo, the toxico-dermas, and finally eczema.

It is a matter of common knowledge that acute urticaria follows the ingestion of certain substances in some individuals. The number of substances capable of producing this eruption is countless. They are for the most part if not always proteins, but W. Jadassohn has shown that it is possible to produce urticaria by means of a pro-

tein-free dialysate. An apparent difficulty at times encountered in attempting to arrive at a diagnosis of the particular substance which produced the reaction is found in the circumstance that the patient may tell us that he has frequently partaken of this substance before without disturbance. An obvious explanation is afforded by what we know of the mechanism of sensitization. It is not the presence of the peccant substance in the alimentary canal that causes the rash. The seat of the disturbance is in the capillaries of the skin which have been sensitized by a previous contact with the substance brought to them through the circulating blood. The process of antibody formation itself occasions no symptoms which can be recognized. The antibodies produced remain fixed in the cells of the capillary walls and a reaction takes place with each succeeding contact of these sensitized cells with the allergen. The harmful substance may have passed through the alimentary canal a hundred times before, but once through some accidental circumstance, it passed more or less unchanged through the intestinal walls into the blood, constituting there a foreign protein. In practice, the problem of determining the cause of the urticaria is complicated by the circumstance that it is often some accidental ingredient of the foodstuff and not the food itself which is the responsible factor. Thus, it may be the presence of some substance in the cow's milk, derived from the food of the animal and not the milk itself which is the antigenic agent. Balyeat narrates a case in which the patient sensitive to peanuts developed a reaction on partaking of milk from a cow that had eaten peanut vine. In the majority of cases of urticaria we have little difficulty in arriving at the probable cause of the disturbance, by closely questioning the patient as to what he had eaten a few hours before the attack began. Often, however, we see the patient after his urticaria has lasted for two or three days and then he may fail to recall the caviar or anchovies with which he commenced his dinner or the few salted almonds he nibbled in the course of it. It is only in those cases in which the urticaria is due to some com-

mon article of diet like egg, milk or bread, which continue indefinitely as chronic urticaria until the allergen is removed from the diet, that we encounter real difficulties. It is in these cases—as well as in other sensitization dermatoses—that we have been in the habit of employing skin tests for diagnostic purposes.

Skin-testing substances and skin tests have been exploited for commercial purposes to such an extent that both the medical and the lay public have come to look upon these tests as an infallible method of ascertaining the cause of an allergic disorder. I have myself been a victim to this delusion, but have now virtually ceased employing them. In my experience skin tests have never given me a clue to the cause of the disorder. As a result of many hundreds of tests made for the most part by experts in this field, I have learned that one patient reacts to pork and to cauliflower—articles of diet of which he never partakes—or that another reacts to a dozen or more different substances, the rigid exclusion of which from his diet brings no visible benefit whatever. I recognize the fact that clinicians occasionally report striking results from skin tests—I have in mind Schloss' classical case of hypersensitiveness to egg (and even in that case the skin test was positive only occasionally)—and that in cases of hay fever and asthma these tests are frequently of the utmost value; but in skin diseases the results are so very rarely of any use and moreover proper carrying out of the tests so tedious and expensive that I do not regard them as worth making. On inquiry among my colleagues, I find that dermatologists in general have come to place very little dependence on these tests. Much confusion exists on this subject which I hope the immunologists will clear up. It is with regret that we must discard a diagnostic procedure so ingeniously conceived and so definite and precise in the response promised.

In chronic urticaria, as in other allergic diseases of alimentary origin, we usually can arrive at a conclusion by inquiry into the history of the case and a careful investigation and control of the diet. The patient should keep a

diary in which he records everything that he eats day by day, and meal by meal, together with a note as to the state of his rash. The repeated coincidence of a particular article of food with an exacerbation or a renewed outbreak of urticaria will provide a clue as to the cause. The definite proof will be afforded by the disappearance of the rash on excluding this article and a fresh outbreak on again partaking of it. Failing to reach a conclusion by this method, it is my practice to put the patient on a rigid vegetable diet restricted to a few articles for eight or ten days. If the rash clears up, it may be assumed that the allergen is an animal protein. To ascertain the particular substance causing the trouble, the patient is then instructed to introduce a single animal protein, such as milk, into his dietary, without making any other change, and if no disturbance follows, to try another, for instance, eggs, the next day, and so on, until a fresh outbreak discloses the cause of the trouble. If no relief follows the exclusion of animal foodstuffs, I reverse the test, put the patient on a strict milk diet and if freedom from the outbreak follows, I introduce each day another vegetable product. In a small percentage of cases of urticaria the harmful substance is not a foodstuff at all, but may be air-borne: Dust from epidermic scales, such as hair, feathers, wool, etc., or pollen. In these cases it requires careful "detective" work to obtain a clue to the cause of the trouble. In a recent article on food allergy, Rowe [1] presents an excellent scheme for testing food allergy by elimination and trial to which I would refer the reader. I have gone into this discussion at some length because the general principles of the determination of the cause of food allergy apply not less to urticaria than to other forms of allergic diseases.

Angioneurotic edema bears such close analogy to urticaria that we need not dwell on it here. It differs mainly in the seat of the sensitized cells. It is not in the superficial vessels as in urticaria but in a larger vascular trunk

[1] Rowe, A. H., *Jour A. M. A.*, Vol. 91, 21 p. 1623, Nov. 24, 1928.

in the hypoderm that the reaction occurs. Angioneurotic edema affects not only the skin but also the mucosa of the alimentary canal and occasionally of the respiratory tract where the acute swelling may constitute a grave menace to life.

Prurigo, both the Hebra type and the prurigo diathésique of Besnier, begins as a pruritus, the most frequent symptom in all forms of skin sensitization. With the occurrence of papular urticaria and wheals the picture is soon changed by the secondary effects of scratching and eczematization or lichenization is produced until we have extensive areas of chronic thickening of all the layers of the skin. There are minor differences between these two forms but they are of no importance for our present discussion. Prurigo is frequently associated with asthma and hay fever.

The group of toxico-dermas which include the drug eruptions, constitutes one of the most interesting forms of allergy. In the first place they afford a clear example of allergy produced by non-protein substances. In the second place, while the drugs differ widely among themselves in chemical constitution—metals, alkaloids, coal tar products, resins, turpentine, halogens, essential oils, etc.—the number of types of skin eruption is limited, so that drugs with the greatest difference in structural constitution produce similar eruptions, and with the exception of the fixed pigmented erythema of antipyrin, phenacetin, phenolphthalein and arsphenamin, and possibly the herpetic eruption of arsenic, no eruption is absolutely characteristic of a particular drug or group of drugs. As with urticaria and angioneurotic edema, the seat of the lesion is in the blood vessels of the skin and though, in a number of drug eruptions, mercury, salvarsan, quinine, etc., the epidermis is involved, this effect is probably only secondary. It is furthermore noteworthy that the mucous membranes are not affected in drug allergy, and that the allergic reaction bears no relation to the ordinary clinical or pharmacological effects of the

drug. The sensitized individual may respond to an infinitesimal amount of the drug. I have seen a quinine rash appear as the result of taking the minute trace of quinine contained in the soda-water-fountain drink called callisaya and soda. Dermatologists rarely have any difficulty in recognizing a drug eruption, but the ignorance of the patient sometimes presents an obstacle to the diagnosis. He may deny emphatically that he has taken any drugs whatever, forgetting the laxative tablet containing phenolphthalein, or the remedy to "break a cold," containing salicylates or quinine which he has been in the habit of taking. It may be mentioned that the pustular lesions of iodine and bromine are not examples of allergy. They are due to the direct action of the drug on the pilo-sebaceous apparatus through which they are in part excreted.

Eczema, constituting about one-fifth of all cases seen in dermatological practice, is the most important of the skin diseases with which we have to deal. For more than a century dermatologists have been struggling with the idea of some peculiar constitution of the blood, a diathesis or a metabolic disturbance, which makes the occurrence of eczema possible. Even to-day, that idea has not entirely been abandoned, and much valuable time and effort is wasted in research on the chemistry of the blood in relation to eczema. With the coming of the bacteriological era, some forty-five years ago, many forms of eczema were recognized as due to the effects of micro-organisms in the skin. A serious obstacle in the way of progress in this field has been the attempt to separate eczemas of known causation from those whose origin was unknown, thereby, as Bloch has said, making our ignorance the basis of classification. A disorder of the skin characterized clinically by redness, swelling, itching and vesiculation, and histologically by signs of inflammation in the upper corium, together with acanthosis, parakeratosis and spongiosis in the epidermis was called eczema when we did not know what caused it; but if it was due to Rhus, primrose, wheat flour, furs, formalin, or other chemicals—if its cause was known—we called it dermatitis. There

is no reasonable basis for this distinction. In my opinion eczema is due either to the direct action of bacteria and fungi, or it is a manifestation of allergy.

In an elaborate series of experiments, Bloch has shown that the skin of the eczematic differs from that of the normal individual in that it is hypersensitive to a variety of test substances, *e.g.*, quinine, formalin, mercury, primrose—to a far greater extent than is the skin of non-eczematic individuals. Expressed numerically, seven eczematics reacted to one or more of these substances, where one normal individual gave a reaction, and, moreover, the eczematic usually reacted to several of the test substances whereas the normal skin seldom reacted, if at all, to more than one. Eczema occurs when an eczematogenous substance comes in contact from without, or by way of the blood stream, with a skin sensitized to that substance.

Contact with primrose provokes an eczema in very few individuals. Bloch has succeeded in obtaining a crystallizable, non-nitrogenous substance from primrose leaves, of which a concentrated ethereal solution provokes an eczema when applied to the skin of any individual, and thereafter that individual probably through life, certainly for a number of years, remains hypersensitive to primrose leaves, contact with which provokes an eczema not only at the site originally treated, but over the entire cutaneous surface. We have in this experiment a reproduction of the process that is puzzling to the layman and often to the physician. The baker with an eczema from flour, the lady with an eczema from a hair dye, will object to our diagnosis on the ground that contact with these substances is not new, but has been going on for years without producing any disturbance. It is precisely the prolonged and intensive contact with these substances that has finally resulted in their absorption and the production of hypersensitiveness.

As might be inferred from the frequency of eczema, the number of eczematogenous substances is enormous. While some of these substances are proteins, the majority prob-

ably are non-proteins, just as it is the non-proteins which produce drug eruptions. Some of these substances produce eczema in a large number of people, some in a few, and some but rarely. Whether a given substance will provoke an eczema, an urticaria, or a toxico-derma depends probably on the particular anatomical element in the skin in which the corresponding antibodies have been fixed.

In the clinical treatment of sensitization-eczema it is, of course, of fundamental importance that the exciting agent be found. Unfortunately we are successful in our search in only a small percentage of these cases. On the other hand it is a fact that we cure a great many of them. As I look back over a dermatological experience of forty years I recall innumerable cases of eczema which have been cured by the remedies at our disposal, without regard to a possible allergic cause for the disease. This fact suggests the possibility that sensitization of cutaneous tissues may not be permanent and that it may be destroyed by the treatment employed, especially by the more powerful reducing agents which we have long regarded as exercising an influence on the deeper tissues of the skin, and above all by the Roentgen rays. I offer this hypothesis with considerable hesitation, unsupported as it is by experimental evidence, though fortified by daily clinical experience.

In practice it is often possible to bring about a cure by the complete change of the patient's environment. A patient with a recurrent erythema and edema of the face and hands, in whom hundreds of skin tests gave no results of value, and that baffled every attempt at determining the cause, was completely cured by a month's stay in the Engadin and there has been no recurrence since her return home three months ago. Another, who lived in a country town where she came much in contact with garden flowers is rapidly losing her eczema of fifteen years' duration since she has changed her residence and studiously avoids flowers. A gentleman attacked by an almost universal eczema last spring was entirely cured by a trip to

Europe and remained free while abroad only to experience a recurrence on returning to his home. His rash again disappeared during a month's stay at Atlantic City but on returning to his home six weeks ago the rash recurred. It seemed obvious that the cause of the disturbance was localized in his own home. On my advice he moved to a hotel and in two weeks the rash has virtually disappeared. In this case the probable cause of the trouble was an insecticide which had been sprayed on his bedding, furniture and rugs last spring, traces of which can be removed only with greatest difficulty.

It is manifestly impossible, within the necessary limits of this address, to cover the subject of allergy in skin diseases except in an inadequate manner. I have not touched on the subject of allergy from physical agents—heat, light, cold, etc., which appears to be an established fact, nor have I mentioned the effects of bacterial sensitization in chronic infections. Dermatologists are well acquainted with the forms of skin diseases known as tuberculides and trichophytides, where the cutaneous lesion is modified by the allergic state of the tissues in which the respective organisms may be deposited. I have said nothing of possible relations of the autonomic nervous system nor of the endocrine glands to allergic diseases. Time does not permit.

The doctrine of allergy has thrown a brilliant light on the field of dermatology. The subject is new; a large number of careful observers in all parts of the world are actively engaged in the study of the problems involved. We may look forward hopefully to the day when methods of determining specific allergy are perfected and a great advance will have been made in the treatment of allergic dermatoses.

Discussion

Abraham Walzer: It has indeed been a great pleasure to have the privilege to listen to the speakers to-night. I think we have all been very fortunate in having heard a most comprehensive discussion on the question of hypersensitiveness, as it applies especially to dermatology, by men who are indeed the recognized masters in their respective fields of medicine.

Owing to the magnitude of the subject I shall limit my discussion to only some of the immunological aspects of urticaria and eczema, especially the chronic and recurrent types, because it is in these diseases I believe that many of the conceptions of hypersensitiveness, so ably presented by the speakers to-night, are well demonstrated.

Dr. Coca has stressed the conception that the skin as a shock tissue can respond to more than one type of lesion as a result of an immunological reaction, *i.e.,* the skin will respond to either urticaria or eczema when it reacts to antigen-reagin stimulation. Besides this peculiar tendency of the skin it has another important property—a non-immunological stimulation of the skin may produce a lesion similar to that of an immunological reaction, *i.e.,* local intradermal injections of such drugs as morphine, codeine, histamine, etc., will produce wheals which are clinically and morphologically identical to those wheals produced as the result of immunological stimulation.

We have experimentally produced wheals on practically every type of skin by various external non-specific means, as the local intradermal injections of histamine. These wheals are non-immunological in mechanism. We have also produced wheals by way of the gastro-intestinal tract, *i.e.,* by the process of local passive transfer of sensitivity from a patient who is sensitive to a certain food. From the injection of the serum of this patient into a subject and then feeding that subject the specific offending food a wheal resulted at the injected site. In this way we have produced the wheal which is immunological in mechanism.

We have also produced wheals on many types of skin lesions, as psoriasis, scleroderma, keloids, ichthyosis, skin atrophies, angioneurotic edema, etc. These wheals were also produced by the method of local passive transfer of sensitivity, that is, we injected the serum of certain food sensitive patients into these particular lesions and then fed the subject the offending food. These wheals were all immunological in mechanism. In other words, we have been able to produce wheals in every conceivable type of skin, normal and abnormal, atopic and non-atopic; all types of wheals were clinically similar but not so in mechanism. We must therefore bear in mind that lesions of all urticarias are clinically and morphologically alike; but all urticarias are not alike in mechanism, some being immunological and some non-immunological.

These facts from a practical point of view are very important. Since all urticarias are not atopic, all urticarias should not be exposed to indiscriminate skin testing. It is for this reason, this common tendency of skin testing every urticaria, that an important diagnostic procedure, skin testing for hypersensitiveness, has fallen into disrepute, especially amongst dermatologists.

As regards eczema, specific hypersensitive eczema, I heartily agree with Dr. Coca in his conception of this particular type of eczema—that the fundamental cause of specific hypersensitive eczema lies in the peculiar quality of the skin. As far as we know, all the factors concerned in the atopic eczema, as the reagins, the excitant, and the other factors concerned in the mechanism of the reaction, are all similar both in eczema and in urticaria, as was brought out by Dr. Coca. The only difference therefore must lie in the specific hypersensitiveness of the skin which will under proper stimulation produce an eczema rather than urticaria; and the main etiological factor for this specific type of skin or shock tissue is heredity. We can support this contention that heredity plays a great part in the etiology of the type of atopic eczema by histories obtained in many of these cases. It is a frequent occurrence to

obtain a familial history of hypersensitive skin, which can be traced through two or even three generations.

The interesting feature connected with this type of eczema is that we cannot experimentally reproduce the lesion of eczema even in an eczematous individual; for when testing the skin of a positive case of atopic eczema, we have this specific shock tissue, the eczematous skin, and we have the excitant, and the reagins, still, on testing we get a wheal—not an eczema. This may possibly be explained by the fact that the antigen instead of being deposited in the injected site through natural channels is directly injected into the skin.

We have seen the counterpart of this in our experimental urticaria studies where we were able to produce a certain type of wheal which we designated as Type 2. These wheals we were only able to produce through the gastrointestinal tract, *i.e.*, we passively sensitized a site on a subject and fed that subject the specific offending food. We were never able to obtain this type of wheal on direct testing, again illustrating the fact that in this particular type the urticarial wheal, as in specific eczema, the offending substance had to reach the skin through the natural channels and by natural means through the blood stream, not by local intradermal injection.

There is another question which perhaps should be answered—whether we can dermatologically select these specific atopic eczemas from the whole group of eczemas. I do not think we are yet able to do this, since, up to the present time, no studies of these types of cases along this line have been made.

Many of these patients (atopic eczemas, especially those that are protein sensitive) show or give a history of involvement of other organs with atopic manifestations, *i.e.*, many eczematous cases demonstrated such lesions associated with the skin condition, or gave a history of asthma or hay fever at some time previous. With such additional clinical data a diagnosis of this type of eczema may not be very difficult.

Summarising the evidence that characterizes this specific atopic type of atopic eczema, we have first, hereditary history of sensitivity, either past or present, in the patient or familial; secondly, positive skin tests—only in cases due to foods or other proteins, such as milk. In drug sensitive cases the skin tests are of no value; thirdly, previous history or present involvement of other organs with atopic disturbance, *i.e.*, cases of atopic eczema with associated hay fever, asthma, etc., or a previous history of such diseases in the patient.

CONCLUSIONS: Disregarding parasitic or other eczemas of obvious external origin, every case of real eczema, especially in children with a definite atopic history, should be considered as atopic first until proven the contrary.

CHARLES MALLORY WILLIAMS: The previous speakers have dealt with this subject principally from the point of view of inheritance and positive sensitization. After a few comments and questions I hope to take up a somewhat different aspect. I understand from Dr. Coca that sensitization, or rather this atopic sensitivity, which results in hay fever and asthma in many individuals and in others in acute urticaria is due to the presence in the blood of some substance which he calls reagin, and that development of this substance is inherited somewhat after the manner of a Mendelian dominant, and that there are figures which show this. I would like to know whether this substance is specific. If it is not, I would ask if it is true or demonstrated that such an individual will react to all of the pollens or substances known to produce asthma and hay fever and, if not, what is the explanation? I have not gone into this and would like to know. It is the presence of this substance which causes reaction which occurs in these sensitive people after ingestion of certain articles of food, because the experiments seem to show that many

proteins are absorbed through the intestinal wall unchanged and are found circulating in the blood of normal individuals, and this, without the presence of reagins, will not produce symptoms. In other words, the presence of reagins is the essential factor of the disease. This would be a very essential advance. Urticaria of course is the disease we all think of in this connection and I was very glad to hear Dr. Walzer make the statement that the origins of urticaria are multiple. The disease owes its name to the symptom caused by external contact with stinging nettle which may be a specific immunological reaction but, somehow, I doubt it. The jelly fish will also produce it. The common cause is by ingestion of certain articles of food, notably strawberries and shell fish. That is the line on which urticaria is usually attacked. I think everyone who has had much experience with dermatology has found many individuals will keep on having urticaria in spite of any food which may be given; they may have milk for a week and nothing else and still have urticaria, and boiled rice for a week and still have urticaria. In other words, we must seek further for the cause of urticaria and what we find here, I think, will probably vary in different cases. Of course at the present time we are very fond of invoking the endocrines and say the suprarenals are out of order, which may or may not be the case. I have seen one case of urticaria with angioneurotic symptoms seemingly dependent on a run down condition, for example, a woman had just given birth to a child, was anemic and neurotic; she was not cured by careful regulation of diet but by hearty feeding, outdoor life and getting her in good health. I believe other cases are due to some focus of infection. There are cases reported of urticaria due to syphilis, but these are exceedingly rare. I believe some cases are due to some bacterial toxin just as we find many cases of erythema are undoubtedly toxic in origin.

Dr. Pollitzer paid his respects to the skin tests and I must say I have had the same experience. They never helped me in practice. The climax to this is reached by room dust; what is there less specific than room dust? It

may contain wool, feathers, linen, silk, etc. It may prove general sensitization, but I cannot see what else it will prove.

One remark Dr. Pollitzer made about drug idiosyncrasy brings up a point with regard to taking of drugs. As regards laxatives, I have found that patients do not consider laxatives as drugs nor headache powders as drugs. If prescribed by a physician they are, but otherwise not. The buying of such patent laxatives over the counter is not considered the taking of drugs and in history taking that is an important point to remember.

The question of dermatitis versus eczema is altogether too long to go into. All these cases are dermatitis, and perhaps the best classification is into dermatitis of known cause and of unknown cause. Eczema is a good deal of a scrap basket. In some cases the poison reaches the skin from without but not from within, and in others the poison reaches the skin from within and not from without. That brings us to one other point, *i.e.*, sensitization of the skin by bacterial toxins from a focus of infection. This is a subject that has not been studied to any extent; we are only on the outskirts of it.

There was recently reported at a dermatological society meeting, the case of a man suffering from eczema, who had been the rounds of dermatologists without improvement. He went to a general hospital and there it was found he had a pus kidney which was removed and then the eczema disappeared.

I had a case of chronic eczema for which I could find no cause, but the patient had chronic appendicitis, and after the removal of the appendix there was no recurrence of eczema. In other words, I think the skin is sensitized to many organisms and responds to the flooding of the system with toxins by the production of rashes. This is not as revolutionary as it sounds.

Dr. Pollitzer referred to the occurrence of tuberculides, *i.e.*, eruptions of various forms in patients already suffer-

ing from tuberculosis caused by the lodgement of the tubercle bacillus or of toxin produced by the tubercle bacillus. I believe at the present time it is considered that many of these so-called toxi-tuberculides are not necessarily tuberculous but may be caused by chronic streptococcus infection. This altered reactivity of the skin is best exemplified by syphilis. If a healthy man is inoculated with spirochaetes on the skin, he reacts by development of a primary sore, characterized by certain well known features followed in a few weeks by secondary eruption. That is the reaction of normal skin.

Supposing a man has had syphilis for a year and then is inoculated with spirochaetae. He does not get the same reaction; he does not get a chancre or secondary eruption. The skin has become immune; the skin has become entirely different. Again, wait ten years and you will find he still has spirochaetes in the body; if he be struck on the shin he frequently develops a gumma on the site of the trauma. Normal skin does not react that way, neither does the skin of a patient in the secondary stage. The reaction of the skin is altered by tuberculosis and by syphilis, but the reaction in the two diseases is absolutely different.

Another disease in which I am particularly interested is less common, *i.e.*, systemic trichophytosis. My attention was called to that subject in rather a dramatic fashion. A child was brought to the dispensary who had a dirty, crusted head, with an eruption on the body looking something like scarlet fever or measles, with a fever of 101°, and I had no idea what the child had. On getting the scalp cleaned up it was apparent she had tinea capitis, the severe type known as Kerion. The rash was a secondary rash caused by toxins produced in the scalp and absorbed into the blood. The following week I read Dr. Bloch's article in the *Annales de Dermatologie,* which explained the whole situation. Those cases are not common in this country, because Kerion is not common. I have seen exactly similar eruptions occur in the skin of patients suffering from ringworm of the feet; the rash disappeared

with the disappearance of the original lesion. Dr. McClasson has reported a case of erysipelas-like eruption of the legs occurring with tinea of the feet and not re-appearing when the tinea was cured. I saw a case in which the ringworm organism was demonstrated from the feet and the erysipelatous eruption, which had recurred for years, did not occur after the tinea was cured. He had large erysipelatous swellings on the legs, high temperature and absolute prostration but, unlike erysipelas, the fever would drop and in twelve hours he would be out playing golf. It is to this class of eruptions, not satisfactorily studied but which I think are vitally important, that I wish to call your attention this evening, *i.e.*, eruptions on the skin caused by irritation of previous sensitized skin by bacterial toxins.

DR. COCA: Dr. Williams asked whether reagins are specific. They are. This is the only property by which we could identify the reagins—that they would sensitize the normal skin site to the same substances to which the original individual was sensitive, and furthermore, wherever there was multiple sensitivity we have always been able to demonstrate a multiplicity of reagins to match this state. Also Dr. Williams asked about the specificity of room dust. He doubted it because of it being such a heterogeneous mixture, but, strange to say, we find a specific excitant in room dust which can be to a certain extent freed from other dirt or other soluble or dialysable substances, and the individual sensitive to room dust has circulating reagins that will sensitize the normal skin to the excitant in dust. This excitant cannot be identified with any other known excitant. If there is a dog in the house, the dust does not give a reaction in an individual sensitive to a dog. The excitant in house dust behaves exactly like the pollen excitant; it must be some vegetable substance.

It is the same thing in house dust the world over. In Europe, Australia and Canada people have been found to be sensitive to our house dust extract as well as their own.

I was interested in the case of eczema due apparently to some infection. We know that the atopic shock organs are susceptible to non-specific influences. We have only to recall the instances of asthmatic patients that are affected by various non-specific influences, such as cold air or certain gases, etc. There is a form of asthma which very likely is due to infection of the upper respiratory tract and which is not due to specific sensitization to the bacteria concerned; when these infected foci are cleared out, the asthma disappears. That corresponds exactly to the case of eczema which clears up after an operation upon an infected abdominal organ.

I am grateful to Dr. Walzer for his discussion of my paper. He has the advantage of being able to speak not only as a dermatologist but as an immunologist. Dr. Pollitzer mentioned primrose, especially the experiments with the active substance in this plant by Bloch. Bloch used his experiments against the theory of the inheritance of atopic hypersensitiveness, but that argument is answered at once by the fact that *dermatitis venenata* is not an atopic condition. No doubt primrose has the position in Europe with regard to dermatitis that ivy and sumach have in this country. Contact is evidently necessary for the development of sensitiveness to poison ivy. Eskimos never come in contact with poison ivy and are completely insensitive to strong extracts of poison ivy, but their near relative, the American Indian, is as sensitive to poison ivy as the white people, *i.e.*, 60 per cent. The reason the Europeans are not primarily sensitive to primrose is that they do not come in sufficient contact with the excitant to develop sensitiveness. The primrose excitant evidently is not airborne as is the oleoresin of poison ivy. An individual highly sensitive to poison ivy has only to drive through a district where poison ivy grows and he returns with a dermatitis affecting all exposed parts.

If one wishes to object to the evidence that so clearly points to a hereditary basis of asthma, hay fever and now of specific eczema, one must not assail it with evidence drawn from a different category of specific hypersensitiveness.

SERUM PROTECTION AND SERUM TREATMENT OF POLIOMYELITIS *

(ABSTRACT)

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The pandemic of poliomyelitis of this era which has startled the world may be dated from 1908-1909 and had as forerunner the gradually enlarging epidemics of Norway and Sweden between 1869 and 1905. These earlier outbreaks led to a remarkable series of studies by Medin, Leegard and others, culminating in the brilliant epidemiological investigation of Wickman in 1905, which has become the basis of our present beliefs. The thorough study of Wernstedt of 1913 supplemented and confirmed the views on the mode of infection in poliomyelitis previously put forward by Wickman. We owe, therefore, the determination of the infectious origin of poliomyelitis to Medin and Leegard, and indeed to Caverly, who investigated an isolated epidemic in Vermont in 1894; and we owe the demonstration of the person to person carriage to Wickman, Wernstedt, Frost and their successors.

The knowledge of poliomyelitis as an epidemic disease dates from the investigations just mentioned, but our present far more complete and detailed knowledge of the pathology of the malady rests less upon studies of human cases than upon the successful communication of the disease to animals. This success is due in the first instance to

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Landsteiner, who in 1909 inoculated rhesus monkeys; next to Flexner and Lewis who accomplished monkey to monkey passages; and subsequently to many other investigators in Austria, France, Germany and America.

Present belief, based on the epidemiology of the human affection and upon the experimental disease produced in monkeys, is that the virus of poliomyelitis, as the infective agent is called, enters and leaves the body by way of the upper respiratory mucous membrane, and especially in the secretions of the nasopharynx. The virus has been detected in the secretions and the substance of this membrane, both in man and monkey. Moreover, the monkey, a relatively resistant species, can be infected by merely instilling a virus of sufficient potency into the nares.

The precise path which the virus takes to gain access to the brain and spinal cord from the nasal membrane can be deduced with tolerable certainty. The virus appears not to pass first into the blood, through which it is carried to the nervous system. Much larger quantities of virus injected directly into the blood do not suffice to infect monkeys. On the other hand, there are cogent reasons for believing that the virus ascends along the olfactory nerve filaments which unite the mucous membrane of the nose to the olfactory lobe of the brain. Probably the descent is also by way of these filaments, in the reverse direction. Since there are reasons for believing that the virus reaches not only the brain but also the cerebrospinal fluid in this ascent, it is likely that the movement upward and downward takes place in the lymph channels of the nerves, rather than in the axis cylinders themselves. Once the brain is reached in this movement, the spread of virus may occur by contiguity of nervous tissues; but as the early clinical manifestations are oftener paralyses of the extremities than of the bulb, it is inferred that the cerebrospinal fluid receives the virus early.

However this may be, and whether the virus ascending along the olfactory nerves works its way along the brain to the more vulnerable spinal cord, or is carried there by

the cerebrospinal fluid, it is quite certain that before it can strike injuriously it must overcome certain obstacles to its penetration. We are informed of two sets of obstacles: One, discovered by Amoss and Taylor, resides in the nasal membrane itself; the other, determined by Amoss and myself, is bound up with the anatomical integrity of the choroid plexus and meninges. If one of these obstructive mechanisms fails, the other may still operate effectively; but if both fail, the result is probably infection and paralysis. Between these two extremes there appear to be partial failures of the protective mechanisms, which may be taken to account for that wide, if not yet accurately defined set of clinical manifestations yielding immunity from subclinical and the several clinically abortive forms of poliomyelitis now described.

Since poliomyelitis is an infectious virus disease, from which recovery in whole or in part commonly occurs, it is not surprising to discover, in view of general knowledge of the class of virus diseases, that recovery results from specific immunization in which a rapid production of immune bodies takes place. These immune bodies have been detected in the blood as early as the sixth day of acute illness; and after recovery from the paralytic disease they have been found to persist in the blood in some degree of concentration as long as twenty years. Moreover, they are detectable in the blood of persons with mild and abortive attacks of poliomyelitis and even in so-called normal persons who have been suspected of subclinical immunization. This last class corresponds of course to other recognized forms of subclinical infection, *e.g.,* diphtheria, leading not to actual illness, but to active immunity.

With this knowledge of the clinical pathology of epidemic poliomyelitis before us, we may inquire whether the employment of already made immune bodies is capable of influencing and moderating the experimental or the natural human form of the disease. It is of course apparent that the first step in this inquiry is to determine the effects of the immune bodies as represented by conval-

escent serum on monkeys. The convalescent serum is taken from recovered cases of poliomyelitis in man and monkey. Thus far it has not been found possible to produce an immune serum in any animal other than the monkey. Since the virus of poliomyelitis is distinct from streptococci, it seems quite empirical and futile to employ an antistreptococcic serum for specific therapeutic purposes.

The procedure employed by Lewis, Amoss and myself to determine the specific protective powers possessed by the convalescent serum was, first, to mix virus and serum outside the body and inject the mixture into the brain of monkeys. This mode of inoculation is the severest test of infectivity which can be employed. When suitable combinations of virus and serum are used, no paralysis results. When insufficiently neutralized virus is injected, the usual incubation period of the experimental disease is prolonged, but ultimately paralysis develops. The next procedure is a far more severe test of the potency of the convalescent serum. The virus is injected into the brain of the monkey, followed by the serum injected by lumbar puncture into the cerebrospinal fluid. When the virus is introduced into the brain, it is deposited in injured brain tissue, under conditions favorable to its protection and multiplication. A part always passes into the cerebrospinal fluid and is carried everywhere in the cerebrospinal axis, including, of course, the vulnerable spinal cord. In this experiment it was hoped that the serum placed also in the cerebrospinal fluid would penetrate the nervous tissues, meet and destroy or neutralize the virus.

In practice this result was actually achieved, when optimal relations of virus, serum, and period of successive injection of virus and serum were secured. In brief, then, this test showed that convalescent serum of man and monkey is capable of neutralizing the virus of poliomyelitis, once it is present under conditions suitable for multiplication in the central nervous organs of the monkey.

We have in these experimental observations the basis of specific serum therapy of poliomyelitis in man. As Dr. Aycock is to deal with this topic in detail, I shall confine myself to a few chronological items only. The experimental results obtained by Lewis and myself were published in 1910, and in 1911 Netter of Paris first applied them to the treatment of early cases in man. A far more comprehensive and basic study of the subject was made by Amoss and Chesney in 1916, during the severe epidemic in New York State. They concentrated attention on early paralytic and preparalytic cases. When marked paralysis existed, betokening severe injury to the nervous system, it was not believed that benefit would accrue from the employment of the serum. It has now become the rule to inject the serum before obvious paralysis occurs. This preparalytic stage of poliomyelitis can be detected by means of accurate clinical and laboratory tests.

I shall now leave this important topic to Dr. Aycock whose experience has been extensive, and devote the remainder of my remarks to the subject of convalescent serum prevention of poliomyelitis.

In order that you may have the subject before you in a clear form, I would like to preface these remarks by a few facts bearing on the anatomical and physiological conditions which predetermine the successful employment of the serum for purposes of passive or protective immunization.

We have already learned that the virus of poliomyelitis passes from the nasal mucosa to the nervous system. It is well known that complex chemicals, including protein, do not pass from the blood to the cerebrospinal fluid as long as the meninges and choroid plexus are anatomically intact. These structures constitute a real barrier between the blood and the substance of the brain and spinal cord. But the barrier is very sensitively adjusted, and can quite readily be impaired by artificial means. Injecting so innocent a fluid as Ringer's solution, and the even more innocent cerebrospinal fluid from another monkey of the same

species, into the subarachnoid space, suffices to open the barrier. When a serum is injected by lumbar puncture, a marked aseptic meningitis is produced and the gates are thrown wide open to the constituents of the blood.

The effects of this impairment can be shown by placing on the nasal mucosa a virus in itself incapable of producing poliomyelitis, or injecting into the blood a quantity of virus impotent in the normal monkey. In both instances, infection and paralysis may follow.

However, this anatomical and physiological impairment of the meninges and choroid plexus may be rendered ineffective if convalescent serum is introduced into the cerebrospinal fluid. By this means the action of the virus in the nares and in the blood may be effectively blocked.

Up to the present, you will have observed that the convalescent serum has been brought directly into relation with the nervous organs through contact with the cerebrospinal fluid. The question arises whether the relation can be secured through the serum injected into the blood. From what has already been stated about the choroid plexus meningeal barrier, this is not to be expected normally; but it may well be expected when the meninges are inflamed. Now, in all cases of symptomatic poliomyelitis, no matter how mild, changes are probably to be found in the cerebrospinal fluid. These changes betoken inflammation, and inflammation indicates a broken barrier. Under these circumstances convalescent serum injected into the blood, may pass into the cerebrospinal fluid and act to block an impending virus infection.

Amoss and I had indeed already followed immune serum from the blood into the cerebrospinal fluid under some such conditions as these, and Stewart and I have recently carried the implications of these considerations into practice by determining that convalescent serum introduced into the blood of monkeys will prevent an intracerebral injection of poliomyelitis virus from producing paralysis. It is hardly necessary to point out the significant applica-

tion of this observation. If in time of danger from epidemic poliomyelitis, adequate supplies of human convalescent serum were available, they could doubtless be employed for purposes of passive immunization.

There remains one more matter of practical importance to touch upon. As you will learn from Dr. Aycock's presentation, the specific serum treatment of early poliomyelitis is carried out by injecting the serum into the subarachnoid space and into the blood of patients. It is customary to make a second intraspinal injection when practicable. The question which Stewart and I undertook to answer is whether the effects of the serum so injected passed off quickly or endured for a time. We found that when 2 cubic centimeters of convalescent serum are injected by lumbar puncture, monkeys are protected against intracerebral inoculation of the virus for at least four days. I am, therefore, of the opinion that while the first intraspinal injection is essential, the second may not be so, and that the continued presence of immune serum in the nervous tissues may be sufficiently provided for by the convalescent serum which has been injected into the blood.

SALPINGITIS *

CHARLES E. FARR and ROBERT E. FINDLAY

A detailed study has been made of all the cases of salpingitis admitted to the First Surgical (Cornell) Division of the New York Hospital from January, 1914, to December, 1927, inclusive. The cases number 545. A simple classification has been adopted as follows:

1. Acute
2. Acute exacerbation
3. Chronic
4. Tuberculous

* Delivered before the Section of Surgery, November 3, 1928.

Of all the cases of salpingitis admitted, 545 as above stated, 115 or 21 per cent were acute, 114 or 21 per cent were acute exacerbation, 293 or 54 per cent were chronic, 26 or 5 per cent were tuberculous.

Age

Salpingitis occurred in this series in individuals as young as eleven and as old as sixty. The average age was 29. Four patients were under 16 years of age. Three of them had never menstruated. Even in these, however, it is doubtful if the tubal infection followed on a juvenile vulvo-vaginitis. A careful study of the incidence at various ages reveals no data of significance.

The marital history was investigated but without results; 420 patients claimed the marriage state; 83 were single; 37 were widows and 5 were divorced. It would not be wise to place too much credence in these data.

The occupations of these 545 women were studied carefully but nothing of any great interest was found; 308 women claimed to be house-wives or were engaged in house work; 59 were domestic servants; 36 were factory workers; 10 were actresses; 9 were telephone operators; 7 were dressmakers; 7 were stenographers; 6 were clerks; 4 were school teachers and the others were scattered.

The symptomatology and laboratory signs of salpingitis were next investigated as follows:

Leucocytosis

A leucocytosis was present in practically all cases on admission, the general average count being 14,500 with 80 per cent polymorphonuclears. The highest was 45,000; the lowest 4,500. The highest polymorphonuclear count was 98, and the lowest, 34.

In acute salpingitis the highest count was 45,000; the lowest, 7,000; the average, 17,000, with the polymorphonuclear percentage ranging from 98 to 61, and giving an average of 84.

Acute exacerbation gave a leucocytosis from 36,000 to 8,000, with an average of 17,000. The polymorphonuclear percentage ranged from 95 to 64, with an average of 83.

Chronic cases yielded a leucocytosis ranging from 28,000 to 4,500, with an average of 11,000. The polymorphonuclear percentage ranged from 90 to 34, with an average of 74 per cent.

Tuberculous salpingitis also showed a leucocytosis ranging from 23,000 to 6,000, with an average of 12,500. The polymorphonuclear percentage ranged from 88 to 67, with an average of 79.

From a combined study of these figures it is seen that there is a leucocytosis in practically all instances of salpingitis, even of the chronic and tuberculous type. This leucocytosis seems relatively high for the amount of pyrexia present in the average case. It is also in our opinion somewhat higher than that seen in similar types of appendicitis. The polymorphonuclear count is high in all forms, even the tuberculous.

Fever

It is noteworthy that nearly all salpingitis patients whether acute or chronic have some fever. In our series the highest was 105.2, the lowest 97.6, average 99.5. Average for acute cases was 101.3. For the acute exacerbation the average was 101.1, for the chronic cases the average was 98.1. In the tuberculous types the average was 100. These are all initial temperatures. We believe that a comparison of a similar series of appendicitis in women for corresponding ages would show that salpingitis as a rule gives a higher temperature reaction than appendicitis.

History, Symptoms and Physical Signs

The history, while of the utmost importance in salpingitis, is also extremely hard to obtain accurately. Many of these women are ignorant, many are careless, and not a few are distinctly adverse to giving an accurate history.

It is important to gain the confidence of the patient and by tactful questioning obtain any history which directly or indirectly would indicate an infection of the tubes. In our series the average remote onset was four and one-half months before admission to the hospital, while the duration of the present illness varied from a few hours to three months with an average of twelve and one-half days.

Considerable stress should be laid upon the chief complaint as stated in the history, especially if the history be well taken. It is noteworthy that nearly 44 per cent of all patients complain of their nausea or vomiting or both. Nearly 10 per cent more complain distinctly of indigestion, that is complaints of heaviness in the epigastrium, heartburn, gastric distension, belching of gas and sour material. These complaints strongly simulate peptic ulcer or cholecystitis rather than appendicitis.

An interesting complaint was that of headache, which occurred in 14 per cent of our salpingitis series. This was a distinct complaint and evidently of ovarian origin.

Dysuria was found in only 18 per cent, while urinary frequency was present in only 12 per cent. This is an interesting contrast to what we are generally taught; that dysuria and urinary frequency are cardinal signs of infection of the genital tract.

A history of gonorrhea could be obtained in only 3 per cent of the series. This was almost invariably blamed on the husband.

A complaint of vaginal discharge was found in 64 per cent. In many instances this was but a slight leucorrhea but, of course, a majority had a considerable amount of yellowish discharge, with occasionally a foul odor. It is interesting to note that 36 per cent gave no history or manifestation of vaginal discharge. This again is contrary to the usual teaching.

Dysmenorrhea was present in only 22 per cent and as other pelvic pathology in the same cases could easily account for the dysmenorrhea it could hardly qualify as a

cardinal symptom of salpingitis—indeed irregular menstruation is considerably more frequent, being found in 31 per cent.

The Location of the Complaint of Pain

No points of especial interest or value were found in a careful analysis of this series. The pain was in both lower quadrants in 48 per cent, in the right lower quadrant in 31 per cent, in the left lower quadrant in 17 per cent. It was present also in the back in 32 per cent. An interesting observation was the presence of pain in the epigastrium in 4 per cent. This is quite a marked point in differentiation from appendicitis in which a very great majority have initial pain in the epigastrium. In appendicitis these gastric pains quickly radiate to the right iliac fossa and disappear from the epigastrium, while in salpingitis, if the pain is in the epigastrium, it usually remains there without radiation.

Other interesting factors obtained from the history were vaginal bleeding in 43 cases; chills in 74; the complaint of fever in 73; sweats in 9; menorrhagia in 9; metrorrhagia in 11; occurrence after the menopause in 3, before puberty in 3, during pregnancy in 3, and associated with syphilis in 9.

In only 72 of our series was there a history of immediately preceding operation, miscarriage or delivery. Thus 13 per cent of the series may be puerperal but even in these it was impossible to determine the presence or absence of gonorrhea.

Type of Pain

An attempt was made to elicit from the patients a descriptive word or two as to the type of pain. Nothing of special value was brought out however. The pain was called "cramplike" in 17 per cent. In 6 per cent it was intermittent. It was stated to be distinctly worse on exertion in 8 per cent, and was described as "sharp" in 3 per cent. Painful defecation was mentioned in 2 per cent and

a falling sensation in 1 per cent. Many other descriptive terms were used but not in sufficient numbers to have any clinical significance.

Many other complaints were mentioned such as loss of weight, flatus, hot flashes, diarrhea, green stools, urinary retention, sterility, constipation, hemorrhoids, etc.

Physical Findings

The most important physical sign in salpingitis is abdominal tenderness. This was present in 83 per cent, being present in both lower quadrants 41 per cent; in the right lower quadrant 28 per cent; in the left lower quadrant 16 per cent; the others were scattered. In marked contrast to this and also in very marked contrast to the signs of appendicitis, abdominal rigidity was noted in only 25 per cent in the entire series.

Vaginal examination revealed tenderness in the fornices —bilateral 40 per cent; right fornix 16 per cent; left fornix 11 per cent.

Masses were found by vaginal examination in 38 per cent. These were situated as follows: Both lateral fornices 8 per cent; right fornix 15 per cent; left fornix 11 per cent; cul de sac 7 per cent.

An interesting observation was the presence of abdominal distension in only 9 cases. This is hardly credible and must be due to lack of careful observation. However, it is quite probable that abdominal distension is much less marked in salpingitis than in appendicitis.

Abdominal masses were found in over 8 per cent. This is an important finding as one does not ordinarily expect to feel abdominal masses in salpingitis.

Diagnosis

The diagnosis of salpingitis can be made in a very large percentage of cases with great ease both from the history and the physical signs but a considerable percentage offers

much difficulty. We would emphasize the importance of the history and the fact that it must be taken with great care and with tact in order to obtain anything like a true picture. Fifty-four per cent of our series were easily diagnosed; 13 per cent more were fairly easy; 5 per cent were in doubt, but probably were salpingitis; 14 per cent were difficult, but were classed as salpingitis eventually, while 7 per cent were wrongly diagnosed either through carelessness or indefinite or incomplete histories and physical examinations, or what is far more probable, because the symptoms and physical signs did not give a clear indication of the real pathology.

Differential Diagnosis

The chief difficulties in differential diagnosis are to distinguish salpingitis from appendicitis, ectopic gestation, and cystoma of the ovary. Many other pathological conditions may also simulate salpingitis and are not infrequently a complication of it.

Naturally appendicitis in one of its many forms offers the chief difficulty in diagnosis. In 19 per cent of all our cases this had to be considered, whereas the possibility of ectopic gestation was considered in 7 per cent; cystoma of the ovary in 7 per cent; fibroid of the uterus in 6 per cent; retroversion in 5 per cent. Many other possible complications were also considered. Endometritis and ureteral colic were not uncommon in this series.

Indications for Operation

Eighty per cent of all our cases were operated upon. Of the acute cases 70 per cent were operated upon and of the acute exacerbation, 83 per cent; 82 per cent of the chronic cases were subjected to operation, and all but one of the tuberculous.

From a careful survey of our results and those of other operators it may be stated with considerable surety that acute salpingitis should never be operated upon; that acute exacerbation should not be operated upon; that

chronic salpingitis, except the tuberculous, should be operated upon; and that the tuberculous should never be subjected to surgery until every other therapeutic resource has been exhausted.

Operative Pathology

There was no special enlightenment from the exhaustive study, from the findings at operation, nor the methods of operative surgery; nor did we obtain any great amount of knowledge from the cultures and smears from the tubes. This is readily understood because the tubes become sterile within a few weeks of the onset of the infection.

The question of drainage is quite important. Our records show that drainage is used much more rarely in recent years—the average being 20 per cent for the entire period. It is believed that drainage can be dispensed with in all cases except those with oozing surfaces or with large accumulations of fibrinous exudate.

Involvement of the ovaries was present in a very large proportion of our cases. This caused partial oophorectomy many times. It was rare indeed that both ovaries were removed.

The appendix was removed in 287 instances but only in a small proportion was there a definite acute disease of the appendix. A few showed acute appendicitis in addition to the salpingitis and one was tuberculous. Many other incidental complications were found but none of any special etiological significance. The most common were endometritis, retroversion, fibromyoma and ectopic pregnancy.

Wound healing in general proceeded normally but a moderate infection occurred in 14 cases. General peritonitis was present in 28. A fecal fistula formed in 7 and peritoneal adhesions were noted in 16. Many other incidental complications were noted.

Pulmonary embolism occurred in 2 cases; lobar pneumonia in 2; broncho-pneumonia in 2; pulmonary tuberculosis was present in 4 instances.

There were two deaths on the table from ether narcosis.

Mistakes in Diagnosis

In 70 per cent of our cases salpingitis was correctly diagnosed. In 3 per cent diagnosis was not made, or at least not posted on the chart. In 27 per cent the diagnosis was incorrect. The proper diagnosis in these latter cases should have been: Appendicitis—53; retroversion—18; ectopic gestation—15. In 13 cases the tuberculous nature of the salpingitis was not recognized.

When to Operate

The rules for operation in salpingitis on the Cornell Division of the New York Hospital are as follows:

1. Acute cases should never be operated upon. This, of course, is subject to an exception where there is a reasonable doubt as to the diagnosis, and particularly when appendicitis or ectopic gestation are possibilities. In case of doubt, we usually operate, but frequently if we find very acute salpingitis, we close the abdomen without attempting to remove the tubes. This may cause a later operation but the mortality is much lower.

2. Operation is not indicated even in a general peritonitis following salpingitis. Practically all cases will recover under conservative treatment.

3. If the salpingitis is not a very severe process it is advisable to remove the tubes even though they are acutely inflamed. The only exceptions are in young girls and those who are especially anxious to have children.

4. In acute exacerbation it is unwise to operate just as in the acute cases. It is better to wait five days after the temperature has reached normal.

5. It is wise to remove all chronically inflamed tubes if there are no contra-indications. This is especially true when tubes are closed.

6. Tuberculous tubes in general should be allowed to quiet down and then be given every possible hygienic care before operation is advised.

Mortality

In this series there were 15 deaths, giving a general mortality rate of 2.75 per cent and a post-operative mortality rate of 3.42 per cent. This includes all fatal issues while the patient was still in the hospital. Twelve of the fifteen deaths were in patients operated upon during active infection. This gives a mortality rate of 9.45 per cent for the acute cases. When one considers that the mortality rate in acute appendicitis is about 5 per cent it is readily seen why operation in acute salpingitis is most unwise.

Causes of death were as follows: 9 from general peritonitis; 4 sudden deaths on the operating table (a comparison of one sudden death on the operating table in 1500 cases of acute appendicitis); 1 of intestinal accident obstruction; 1 acute dilatation of the stomach; 1 cerebral; 1 lung abscess.

There were only 22 (5 per cent) secondary operations in this series during the hospital stay. They were almost entirely for the relief of residual abscesses. There was one intestinal obstruction and one ectopic pregnancy. There was one immediate post-operative hernia and one fecal fistula.

The post-operative results are very interesting but our data are not as accurate as could be desired. These women did not coöperate to anything like the extent that other similar groups did in our service.

Three per cent developed post-operative ventral hernia. Nine of these herniae followed abdominal drainage. The

question of post-operative pregnancy was studied as carefully as our records permitted but we are unable to give any light on this subject. Sixteen reported pregnant, but of these three only had living children; 4 developed ectopic gestation; 2 had miscarriages; 6 were too early in pregnancy at the time of examination to be sure of the result. Forty per cent of our series returned and were judged as giving satisfactory results. A further 20 per cent were considered satisfactory but with complaints of a minor character; 18 per cent were not traceable; 13 per cent were re-admitted for one complaint or another, usually of a pelvic nature. Ten per cent were uncoöperative and no post-operative records were made.

Conclusions

Salpingitis is a serious disease. Its diagnosis is not easy and treatment requires much surgical skill and judgment. Acute cases should never be operated upon. There should practically be no mortality in chronic cases. The end results are fairly good, considering the nature of the disease and the many complicating factors.

LUNG ABSCESS

SOME ASPECTS OF ETIOLOGY AND MEDICAL TREATMENT *

James Alexander Miller

Etiology

The generally accepted idea of the etiology of pulmonary suppuration has been that it follows certain cases of pneumonia in which, because of certain conditions in the lung little understood, necrosis and suppuration result instead of the more usual process of resolution.

* Delivered before the Sections of Laryngology and Rhinology, and Medicine, November 28, 1928.

Certain etiological factors have been recognized as especially favoring the development of pulmonary suppuration, such as the inhalation of foreign bodies, operations upon the respiratory tract, especially tonsillectomy and the extraction of teeth, and less frequently following operations upon suppurative foci in more distant parts of the body. In all of these conditions the conception has been that pneumonia first occurred and that then later following the pneumonia there was suppuration.

Dr. Adrian Lambert and I in studies of a considerable series of abscess of the lung were impressed by the fact that a very considerable percentage of the cases apparently occurred without any evidence of either pneumonia or of any of the predisposing factors above mentioned. The fact, also, that in cultures made from these lung abscesses very frequently none of the ordinary pathogenic bacteria could be found, although the abscesses swarmed with anaerobic bacteria, led us to suspect that possibly the anaerobic organisms played a real pathogenic role in the formation of the abscess instead of simply the secondary one which had been ascribed to them.

It is significant also that the anaerobes found in abscesses correspond to a group frequently found in persons with bad oral hygiene, particularly diseased tonsils and pyorrhea alveolaris.

Since that time a considerable amount of experimental evidence concerning the role of anaerobes in the formation of abscesses has been elicited. Klein and Smith have produced pulmonary abscesses in animals by intratracheal injections of the scrapings from infected teeth.

Crowe and Scarf injected the sinuses of dogs with such scrapings and obtained abscesses of the lung in two out of six cases.

Fetteroff and Fox found infected emboli in the bed of removed tonsils.

Cutler and Schlueter were able to produce pulmonary abscesses by placing emboli infected with anaerobes in the jugular vein of animals.

From these experiments it would appear that abscess of the lung may be produced either by inhalation of infection or through the blood stream by embolus from more distant parts of the body. It also appears to be proved that the anaerobic bacteria frequently found in the mouth will produce abscesses of the lung experimentally under certain conditions.

The anaerobes which have been found most commonly, both in dirty mouths and in abscesses of the lungs, are some of the spirochaetes, fusiform bacilli, vibrios, motile bacilli of the colon group and anaerobic streptococci.

The important practical application of these newer ideas of the possible role of the anaerobe in the production of pulmonary abscess is the fact that operative procedures, especially tonsillectomy and tooth extraction in the mouths of persons with poor oral hygiene, produce ideal conditions for the transmission of these organisms to the lungs and point to the extreme importance of cleaning up the mouth very carefully prior to any such operation. It would appear that as a preventive procedure this is most important and is probably more so than the much discussed problem of the relative danger of local and general anaesthesia for such operations.

Medical Treatment

Early abscess of the lung should be considered primarily as a medical disease. Chronic pulmonary abscess usually calls for surgery.

Rest and Posture

It is our experience that rest, combined with proper postural treatment, is the best method to be employed first in the treatment of early pulmonary abscess. From forty-five to fifty per cent of early abscesses recover by this means alone. It has long been known that a certain number of abscesses of the lung cure themselves completely by spontaneous rupture and evacuation. It is the object

of this medical treatment by rest and posture to increase the number of such cures and the advantage to the patient of avoiding operation with a very long convalescence and frequent surgical dressings, to say nothing of the danger of the operation itself, is very evident.

We have also found that surgical operation during the acute phases of abscess of the lung carries with it a very high mortality, sixty-five to seventy per cent. By careful frequent x-ray examinations we believe it is possible to gauge the point when this acute inflammation about the abscess is subsiding and when such x-ray studies are associated with frequent surgical consultation, as they should be, it is usually possible to judge of the phase of the disease in which operation can be performed with the least risk and with the best hope of success.

On the other hand, if the lung lesion does not clear up satisfactorily after one or two months of this conservative treatment, we have found that it probably will not and that then surgery should be called upon. If this medical treatment is persisted in too long, the case drifts into the chronic stage and operation then again becomes more dangerous and complete cure is less apt to occur because of the liability to permanent bronchial fistula or to the failure of the abscess to close completely on account of the rigid fibrous walls which have been allowed to form.

Medical care, therefore, consists mainly in insisting upon rest and postural drainage during the early acute stages, and later consists in the exercise of proper judgment as to whether this treatment alone is likely to succeed, rather than to allow the case to become too chronic and less favorable for the surgical operation which will eventually be necessary.

Bronchoscopy

In association with rest and posture, we have used bronchoscopy as an aid in the treatment of certain cases in order to assist drainage. We usually employ this method in cases which do not respond in a few weeks to

rest and posture alone before resorting to more radical surgery. When foreign-body inhalation is suspected or possible, bronchoscopy is indispensable both for the diagnosis and for the cure by removal of the foreign body.

On the other hand, we believe that the practice of long continued bronchoscopy over many months is mainly palliative and has the same unfavorable effect as does the long-continued medical treatment, allowing the case to become so chronic as to be surgically unfavorable.

Artificial Pneumothorax

Artificial pneumothorax has been advocated by some as an ideal method of treatment for abscesses of the lung. We have used this method in a considerable number of cases and found it very unsatisfactory and sometimes dangerous. If the abscess of the lung is situated near the periphery the introduction of air into the pleural cavity may result in the tearing of adhesions, with the rupture of the abscess into the pleural cavity and the occurrence of a very fulminating empyema, which is generally fatal.

On the other hand, the air introduced by the method of artificial pneumothorax can not be exactly controlled and sometimes it will get in between the lung and the mediastinal pleura, resulting in a cutting off of the drainage rather than in favoring it. We believe that the principle of artificial pneumothorax as it is used in pulmonary tuberculosis is mainly for immobilization of the infected organ. The principle of treatment in pulmonary abscess, on the other hand, is drainage, which is by no means always favored by this method.

Summary

1. Abscess of the lung is frequently caused by the post-operative infection from infected mouths.

2. Cleanliness of the mouth is a very important prophylactic measure in avoiding post-operative pulmonary abscess.

3. The diagnosis of pulmonary abscess is comparatively simple and depends largely upon x-ray evidence.

4. The treatment of acute pulmonary abscess is primarily medical by rest and posture.

5. Bronchoscopy is a valuable aid in some cases and is indispensable in cases due to the inhalation of foreign bodies.

6. Medical treatment and bronchoscopy should not be continued so long that the case becomes chronic.

7. Surgery is necessary in about fifty per cent of the cases and if employed at the favorable stage is not attended with serious risk. It is usually the only treatment for chronic cases.

8. The close coöperation of the nose and throat surgeon, the internist, the surgeon and the bronchoscopist is essential to obtain the best results.

EXCERPTS FROM THE MINUTES OF THE COUNCIL

JANUARY 30, 1929

The Trustees reported that the expenses of the Academy for 1928 were $259,465.93 and the income $267,846.19; and that the balance of $8,380.26, consisting of $6,204.63 of restricted funds for poliomyelitis, etc., and $2,175.63 of unrestricted surplus, had been added to the Furniture and Fixture Reserve. The land, building and contents is valued at $2,560,070.97. The total of the various funds amounts to: $1,958,009.92

Total invested	1,934.696.25
Balance uninvested	23,313.67
Possible tax refund.....	6,542.24
Streeter pledges	13,443.00
Cash	3,328.43
	23,313.67
Income account invested.....	55,460.37

There has been received from the Rockefeller Foundation $500,000 account principal and $12,500 account income for 1929. With the advice of the Advisory Finance Committee the sum of $563,287.50 has been invested.

The report of the Library Committee was presented and it was moved and duly carried that the Council approves of publishing in the By-Laws and in the Rules and Regulations of the Library that Library subscription fees shall be $40 for individuals and $100 for commercial firms. It was also

Resolved, that the Council of the Academy learns with great satisfaction of the interest which has been taken in our incunabula by Dr. Arnold Klebs of Nyon, Switzerland, and hereby expresses to Dr. Klebs its very deep appreciation of the valuable services which he has rendered this institution. It was also

Resolved, that the Council hereby expresses its appreciation and thankfulness to Dr. Frank C. Hollister for his donation of the Argellatta incunabula.

It was moved and duly carried that the Council is still in favor of affiliation between the Pathological Society and the Academy being consummated.

It was also moved and duly carried that an invitation be extended to the Society for Experimental Biology and Medicine to use one of the Academy meeting rooms on the third Wednesday of the months of October to May inclusive with lantern and operator free of charge.

It was also moved and duly carried that the hospitality of the Academy be offered to delegates to the International Hospital Congress and that they be informed that they may use the Academy as a mailing address. It was

Resolved, that the Council feels that no one except a Fellow of the Academy should enter into discussion at any meeting of the Academy or its Sections unless especially invited to do so and that this matter be referred to the By-Laws Committee for consideration.

THE LIBRARY

Recent Valuable Accessions

During the last few weeks some valuable books have been received in the Library. Two were purchased through the Rare Book Fund, one being an Avicenna manuscript of the XIIIth century, the other, the British Museum *Catalogue of XVth century books* in six volumes. The Avicenna although imperfect, is one of the oldest manuscripts we possess. It is a Latin translation of the second and third books of the *Canon* which was the medical authority of the Middle Ages. The British Museum *Catalogue* is very difficult to acquire complete and we are especially fortunate in securing it at this time as it is of invaluable assistance in the cataloguing of our incunabula.

Our incunabula continue to grow in number. Dr. F. C. Hollister presented to the Library: Petrus de Argelatta, *Libri sex chirurgiae,* Venice: Benedictus Locattellus for Octavianus Scotus 1497. 8 Kal. Mar. [Feb. 22]. It is a very good copy with the De Vinne book-plate. Dr. Hollister also gave us an autograph letter, signed, of Sir James Y. Simpson.

Another gift from a Fellow, Dr. F. C. Matthews, was Fabricius *De venarum osteolis,* 1603. Although not the first edition, it is an early one and in very good condition with excellent plates. This is one of the works which led Harvey towards his far-reaching discovery of the circulation.

Dr. Howard Lilienthal has given us several important books, amongst which are the minutes in manuscript, of the meetings of the Physico-medical Society of New York, from 1815-1924. It seems very fitting that it is now in the Academy. This group of books belonged to Dr. Joseph Mather Smith who was President of the Academy in 1854.

Recent Accessions

Aufrecht, H. Kenntnis und Behandlung innerer Krankheiten.
Breslau, Priebatsch, 1928, 192 p.

Celli, A. Die Malaria in ihrer Bedeutung für die Geschichte Roms und der römischen Campagna.
Leipzig, Thieme, 1929, 117 p.

Collier, W. A. & Cohn, A. Mikroskopischer Nachweis der Spirochaeta pallida. . .
Berlin, Urban, 1928, 92 p.

Cushing, H. W. & Bailey, P. Tumors arising from the blood-vessels of the brain.
Springfield, Ill., Thomas, 1928, 219 p.

Dible, J. H. Recent advances in bacteriology. . .
Phil., Blakiston, 1929, 363 p.
Fabian, H. Spezielle Anatomie des Gebisses.
Leipzig, Klinkhardt, 1928, 131 p.
Fowweather, F. S. A handbook of clinical chemical pathology.
Phil., Blakiston, 1929, 216 p.
Garrison, F. H. An introduction to the history of medicine.
4. ed., Phil., Saunders, 1929, 996 p.
Gershenfeld, L. Bacteriology and sanitary science.
Phil., Lea, 1929, 432 p.
Handbook of microscopical technique. . . Edited by C. E. McClung.
N. Y., Hoeber, 1929, 495 p.
Herrmannsdorfer, M. & Herrmannsdorfer, A. Praktische Anleitung zur kochsalzfreien Ernährung Tuberkulöser.
Leipzig, Barth, 1929, 49 p.
Hoche, A. Schlaf und Traum.
Berlin, Ullstein, [1928], 149 p.
Hygiene und soziale Hygiene in Hamburg. . .
Hamburg, Hartung, 1928, 672 p.
Jelliffe, S. E. & White, W. A. Diseases of the nervous system.
5. ed. Phil., Lea, 1929, 1174 p.
Kaufmann, E. Pathology for students and practitioners.
Phil., Blakiston, [1929], 3 v.
Lehrbuch der Toxikologie. . . Hrsg. von F. Flury und H. Zangger.
Berlin, Springer, 1928, 500 p.
Morse, J. L.; Wyman, E. T. & Hill, L. W. The infant and young child.
2. ed., Phil., Saunders, 1929, 299 p.
Olivares, L. Fracturas.
Madrid, Morata, 1928, v. 1.
Owens, C. E. Principles of plant pathology.
N. Y., Wiley, 1928, 629 p.
Petermann, B. Die Wertheimer-Koffka-Köhlersche Gestalttheorie und das Gestaltproblem.
Leipzig, Barth, 1929, 292 p.
Ritchie, A. D. The comparative physiology of muscular tissue.
Cambridge, University Press, 1928, 111 p.
St. Mary's hospital, Rochester, Minnesota. The operating room.
2. ed., Phil., Saunders, 1928, 235 p.
Stoss, A. O. Tierärztliche Geburtskunde und Gynäkologie.
Stuttgart, Enke, 1928, 432 p.
Stratz, C. H. Der Körper des Kindes und seine Pflege.
11. Aufl., Stuttgart, Enke, 1928, 398 p.
de Vecchis, B. Manuale di odontoiatria e protesi dentaria per medici e studenti.
Torino, Unione Tipografico-Editrice Torinese, 1928, 670 p.

CATALOGUE
OF
AN EXHIBITION OF MEDICAL MANUSCRIPTS AND INCUNABULA

An exhibition of some of our oldest manuscripts and medical incunabula (books printed before 1501) was set up in the Library and ready for the meetings of the History of Science Society which were held on 28th and 29th December. Many of the books came to the Academy with the E. C. Streeter collection which was purchased by the Academy in the spring of 1928, the rest of the volumes were already in our possession. Altogether we have about one hundred and thirty incunabula but we could not show them all in the exhibition cases which were available. Dr. E. C. Streeter, of Boston, himself together with Dr. Arnold C. Klebs, of Nyon, Switzerland, are largely responsible for the choice which was made for the exhibition. Those which were shown are worthy of notice as examples of the printer's art or because they are editions of medical works which exerted a strong influence at the time they were published. We are deeply indebted to these gentlemen for their help. Dr. Klebs is a widely acknowledged authority on early printing and the history of medicine and we are reaping the benefit for he is very kindly cataloguing all our incunabula.

The exhibition is being kept open throughout the months of January and February. The entries, copied from the cards placed in the exhibition cases with the books, are arranged chronologically.

MANUSCRIPTS

Guilielmus de Saliceto (of Piacenza).
La cirurgia.
Italian Manuscript, dated Bologna 1268, with which is bound:

Guilielmus Parmensis.
Cirurgia and other tractates.
Latin Manuscript, dated 1469.
(Francisc. de Senis, de balneis. Petras de Abano. tract. de venenis. Arn. de Villanova. De arte cognoscendi venena.)

Avicenna (Ibn Sina).
Canonis libri ii et iii interprete Gerardo Cremonensi.
Manuscript in Latin, Italy, end of XIIIth century.
Avicenna or Ibn Sina (980-1036) of Bagdad was court physician and vizier to different caliphs. The *Canon* was an attempt to codify all medical knowledge and to show how it fitted in with the systems of Aristotle and Galen. It was the authority in the Middle Ages.

Serapion (Junior).
Liber de medicinis simplicibus.
Manuscript, Italy, early XIVth century.

Collection of alchemy, philosophy, etc.
Manuscript. ? Italy, XIVth century.
Opened at St. Thomas Aquinas 'De motu cordis'.

Arnoldus de Villanova.
Aphorismi medici.
Latin Manuscript XVth century.

Guy de Chauliac.
Inventorie or the Collectorye in Cirurgicale parte of medicene . . . .
Manuscript in English, late XIVth century or early XVth.
The author (1300-1368), of Avignon, was the most distinguished authority on surgery in the XIVth and XVth centuries. The only other known MS. in English is in Paris. Possibly the copy was made for John of Gaunt. It would be a treasure in any library. Besides the illuminations there are illustrations in the text.

Anonymous Latin Manuscript.
Early XVth century.
The incipit states that "here are given prescriptions made out according to my experience [word erased] from [word erased] to 1424 month of December after the ninth book of Almansor". It is therefore a commentary upon Rhazes' popular textbook.

INCUNABULA

Nicolaus Salernitanus.
Tractatulus quid pro quo and Sinonima.
Venice, Nicolaus Jenson, 1471.
The author flourished about 1100. The antidotarium should precede the two little tracts contained in this fragment. The *Sinonima* is a dictionary of simples.

Petrus Lombardus.
Liber sententiarum.
Strassburg, Heinrich Eggestein, not after 1471.

Petrus Lombardus (? 1100-1164) was a celebrated Italian theologian whose fame rests upon this book of moral precepts which for many years enjoyed a tremendous vogue.

Metlinger, Bartholomaeus.
Ein Regiment der jungen Kinder.
Augsburg, J. Bämler, 1474.
This is the second book on diseases of children and was printed two years after the *Libellus de egritudinibus infantium* of Bagellardus. It is a compilation which absorbed practically all of Bagellardus.

Aristotle.
De animalibus libri.
Venice, J. de Colonia and J. Manthen, 1476.
Aristotle (384-322 B. C.) is, after Hippocrates, the greatest name in early medicine. He gave to medicine the beginnings of zoology, comparative anatomy and embryology. His works were the sources of almost all philosophical and scientific thought throughout the Middle Ages. This is considered by Dibdin to be one of the most beautiful volumes from the press of these printers.

Ortolff von Beyrlandt.
Arzneibuch.
Nuremberg, Anton Koberger, 1477.
Several editions of this work appeared between 1477 and 1491. The author, a XVth century physician, published also a popular work on gynecology.

Benedictus de Nursia.
De conservatione sanitatis.
Thaddaeus de Florentia.
De regimine sanitatis.
Bologna, Dominicus de Lapis, 1477.
Benedictus, professor at Perugia, physician to the pope and to the Sforzas of Milan, attained to high papal and state honors and died in 1450. The first edition of this work was published at Rome in 1475. Thaddeus Alderotti (died 1303) was 'the Galen of his time'.

Conrad von Megenberg.
Buch der Natur.
Augsburg, Johann Bämler, 1478.
Conrad von Megenberg (? 1309-1374), a scholar-priest, wrote this book in 1349-50. It is based on an earlier work by Thomas Cantimpré and was very popular even after the invention of printing for there were six Augsburg editions before 1500. It is a compendium of natural history including medicine and is illustrated with wood cuts. This is one of five known copies of this edition.

Celsus, Aulus Cornelius.

De medicina.

Florence, Nicolaus Laurentius, 1478.

Celsus lived in the Augustan age and, though probably not a medical man, wrote this book on the state of medicine of his day, and a good outline of the history of medicine up to his time. He was unknown throughout the entire Middle Ages but was rediscovered in 1300 by Simon Januensis. This copy belonged to Dr. Hartmann Schedel, the humanist.

Mesue, Johannes, Junior.

Opera.

Pavia, Franciscus de Sancto Petro, 1478.

A Latin compilation by 'Mesue Junior' appeared in the Xth or XIth century but the Arabic originals have not been found. Probably the author's name is a pseudonym. This is the only copy listed of this edition.

Albertus Magnus.

De animalibus.

Mantua, Paulus de Butzbach, 1479.

Albertus Magnus (1193-1280) (Albert von Bollstädt) monk, theologian, scholastic philosopher, teacher at Paris and Cologne, and Bishop of Ratisbon, was a prolific writer on many subjects but was most concerned with the study of nature. Many of his writings dealt with medical subjects. *De animalibus* first appeared in Rome in 1478.

Mensa Philosophica.

Cologne, Conrad Winters de Homborch, 1479-80.

This book is attributed to Michael Scott, one of the greatest XIIIth century philosophers. There are chapters on the appetite, food, drink, baths, women, etc. At the court of Frederic II of Sicily he did much to further the study of natural sciences and medicine.

Matthaeus Sylvaticus.

Liber pandectarum medicinae.

Venice, Johannes de Colonia and Johannes Manthen, 1480.

The author, born at Mantua, died in 1342(?). This book, an early attempt to reconcile the schools of Arabian and Greek medicine, first appeared in 1474. It arranges the drugs alphabetically according to the Arabic names and gives the Latin equivalents.

Guilielmus de Saliceto.

De salute corporis.

Johannes de Turrecremata.

De salute animae.

[Cologne, Cornelis de Zierickzee ab. 1490.]

Saliceto (?1210-1277), professor at Bologna, was the ablest Italian surgeon of his century. This book is a semi-popular guide to healthy

living. Johannes de Turrecremata, a powerful prince of the church in Rome, wrote the second tract for the salvation of the soul. It was exceedingly popular throughout the Renaissance.

Guainerius, Antonius.
Opera medica.
Pavia, Antonius de Carcano, 1481.
Antonio Guaineri was a member of the famous faculty of Pavia and a colleague of Gianmatteo da Gradi. Like the latter he represented the pure Arabic and scholastic trend of learning before the advent of the Renaissance. He merits study, however, as his clinical observations, notably those on aphasia, are very shrewd.

Dondis, Jacobus de.
Aggregator.
Venice, Michele Manzolo, 1481.
The author (1298-1359), "citizen of Padua", was a mathematician and horologist. The book is the first great dictionary of treatment and gives the drugs and simples under Greek, Latin and Arabic names.

Abraham Ben-'Ezra.
De luminaribus et diebus criticis.
Padua, Matthaeus Cerdonis, 1482.
The author (?1092-1167), a Spanish Jew, was a philosopher and astronomer. This astrological book is about critical days in disease.

Bartholomaeus Anglicus.
De proprietatibus rerum.
Nuremberg, Anton Koberger, 1483.
The author, an English Franciscan monk, flourished 1230-1250. This work is an encyclopaedia of which books 5, 6 and 7 are medical. Note the interesting initial letter done by hand.

Arnoldus de Villanova.
Breviarium practicae.
Milan, Christophorus Valdarfer, 1483.
Arnoldus de Villanova (1253-1312) was a doctor of theology, law, philosophy and medicine. He made some independent observations. This book was one of the best medieval handbooks.

Opusculum repertorii pronosticon et Hippocratis libellus de medicorum astrologie.
Venice, Erhard Ratdolt, 1485.
These collected treatises, some of which were erroneously attributed to Hippocrates, contain the astrological lore of the times. The knowledge of starcraft was part and parcel of the training of every physician. The printer of this pamphlet, Ratdolt, a German of Augsburg, was one of the greatest and most artistic printers of Venice. He was the first to print in several colors.

Simon a Cordo Januensis.
Clavis sanationis.

Venice, Guilielmus de Tridino of Monteferrat, 1486.

Simon a Cordo of Genoa, the translator (with the help of a Jewish scholar) of the Arab treatise on simples by Serapion, is of importance as one of the first to improve medical nomenclature. He brought order into the linguistic chaos of medieval translators. His *Clavis sanationis*, or medical synonyma, gives the results of his labours in the field of pharmacology. He was papal archiater to Nicolas IV.

Vincentius Bellovacensis.
Speculum naturale.

[Strassburg, Printer of Legenda aurea, ab. 1480.]

Vincent of Beauvais (died 1264) was a learned Dominican, a contemporary of Saint Louis. He produced the most exhaustive encyclopaedic work of his time on the extant knowledge of the world and its objects, the *Speculum majus*. Two parts of this book, the *Speculum naturale*, and the *Doctrinale* contain medical information.

Avicenna (Ibn Sina).
Lib. Canon. V. et libellus de viribus cordis.

Venice, Petrus Maufer et socii, 1486.

From the early XIth through to the XVIIIth century Avicenna was usually referred to as the "Prince". As such he ruled as the supreme authority in medicine. He was the great assimilator of Greek science and great also in systematizing empirical knowledge. His *Canon*, extant in numerous MSS. and editions, is fully as important as Galen's earlier work.

Savonarola, Iohannes Michael.
Canonica de febribus.

Bologna, Dyonisius de Bertochis, 1487.

Iohannes Michael Savonarola (1390-1462), grandfather of the famous Florentine reformer, worked mostly in Ferrara. His clinical work marks a distinct departure from traditional scholastic teaching. He wrote various treatises which enjoyed great vogue.

Articella or Thesaurus operum medicorum antiquorum.

Venice, Baptista de Tortis, 1487.

Contains works by Joannitius (Hunain ibn Ishaq), Philaretus, Theophilus, Hippocrates, Galen, Gentile de Fulgineo.

Ficinus, Marsilius.
De vita sana.

Florence, Ant. Mischominus, 1489.

This Florentine physician was also a member of the Platonic Academy. His treatise on the healthy life had an immense popularity in Europe.

Maimonides, Moses.

Aphorismi medici.

Bologna, B. Hectoris, 1489.

The *Aphorisms* of Maimonides (1135-1204) are chiefly abstracts from Galen and other ancient authors, but there are many modifications based on personal experience. Maimonides is also notable as an early critic of Galen.

Dinus del Garbo.

Compilatio emplastrorum.

Ferrara, Andreas Bellfortis, 1489.

Dino del Garbo, one of the eminent "expositors" of Avicenna, belongs to the school of Bologna. He came from Florence where he died in 1327. Though often accused of plagiarism, he was physician to King Robert of Sicily and commanded high fees.

Guilielmus de Saliceto.

Cirurgia.

Venice, ? printer, 1489.

William of Piacenza (Salicetus) was the greatest surgeon of the XIIIth century. He furthered the scientific side of surgery and technically is noted for having brought into practice the skilful use of the knife as opposed to cautery. He is also remembered for effectively fighting against the separation of surgery from medicine.

Lanfrancus, Mediolanus.

La cirurgie. Translated into French by Guillaume Yvoire.

Lyons, Jean de la Fontayne, 1490.

This is a very rare French edition of Lanfranc's treatise on surgery. Lanfranc belongs to the school of William of Saliceto. He also is Italian and has the great merit of having introduced the scientific methods of his master to the Collège de St. Cosme in Paris, thereby laying the foundation for the later brilliant activities of French surgeons.

Louffenburg, Heinrich von.

Versehung des Leibs.

Augsburg, ? Erhard Ratdolt, 1491.

Heinrich von Louffenburg's book is a German regimen of health (1429) in rhyme, with quaint woodcuts. The present edition was printed at Augsburg and is probably one of the rarest of the popular medical books. Only two other copies besides this one are registered.

Falcutius, Nicolaus.

Sermo tertius de membris capitis.

Venice, Bernardinus Stagninus de Tridino of Monteferrat, 1490.

Nicolo Falcucci, the Florentine (often confounded with his namesake of Salerno), died early in the XVth century. His great medical opus, the *Sermones medicinales*, is an elaborate compilation which brought fame to its author, in spite of the fact that it contained few original observations.

Arnoldus de Villanova.

Speculum medicinae. (ed. by Martinus Mellerstat).

Leipzig, [Lotter, 149—.]

Arnoldus, a shining light in medicine of the XIIIth century, taught mainly at Montpellier, though also at Naples and Paris. A good clinician and physician, his services were in demand by Pope and King.

Gazius, Antonius.

Corona florida. De conservatione sanitatis.

Venice. Ioh. de Forli & Gregorius fratres. 1491. June 20.

Gazio lived (1469-1530) in Padua and other Italian cities as a much sought-after physician. His *Corona florida*, a regimen of health, is based chiefly on Arabic tradition. It was very popular.

Hugo Senensis (Ugone Benzi of Siena).

Expositio in I. fen Primi Canonis Avicennae. Ant. Faventinus. De febre.

Ferrara, Andreas Belfortis Gallus, 1491. Aug. 13, with which is bound:

Barzizius, Christoforus.

Introductorium sive Ianua ad opus practicum.

Pavia. Antonius de Carcano, 1494, Aug. 20.

Ugone Benzi of Siena was one of the best known medical teachers and clinicians. He taught mostly in Padua where in 1429 he is known to have dissected the body of a criminal. Barzizi, whose chief work is bound also in this volume, was renowned for his collection of *Consilia*.

John of Gaddesden.

Rosa medicinae.

Pavia, Johannes Ant. Birreta, 1492.

The author of the "Rose of Medicine" John the Englishman, (about 1280-1361), was in many ways the typical medieval scholar, who borrowed abundantly from the Arabs. His recommendation to his confrères, to collect their fees before the cure, and his use of red cloth for the avoidance of pox marks have, however, a modern flavor. The "Rose" followed and imitated Bernard of Gordon's "Lily".

Leonicenus, Nicolaus.

Plinii ac aliorum de simplicibus medicaminibus errores.

Ferrara. Laurentius de Rubeis & Andreas Grassis de Castronovo socii. 1492, Dec. 18.

A humanistic pamphlet in the fight for and against Pliny the Elder. The letter which serves as preface, is by Angelo Poliziano, the famous poet and member of the Platonic Academy in Florence, who with his colleagues was most active and enthusiastic over the newly discovered truths of the ancients.

Collenucius, Pandolphus.

Pliniana defensio adversus accusationem Nicolai Leoniceni.

Ferrara. Andreas Belfortis Gallus. ? 1493.

Pandolfo Collenuci of Pesaro (died 1504) attacked the calumniators of Pliny the Elder. He was a conservative rather than a critical modern like Nicolaus Leonicenus and Hermolaus Barbarus, who were fighting against the tyrannical authority of the ancients.

Celsus, Aulus Cornelius.
De medicina lib. VIII.
Venice, Ioh. Rubeus. 1493, July 8.
Though probably not a practising physician, Celsus has contributed one of the best balanced medical treatises that have come down to us from Galen's time. He was not discovered until the Renaissance and so was entirely unknown during the Middle Ages.

Mundinus.
Anathomia.
[Leipzig, Martin Landsberg, ab. 1493.]
Mundino de' Luzzi (born about 1275), son of an apothecary of Bologna, wrote this little treatise of systematic anatomical investigation based on autopsies. For three hundred years it was the most important anatomical primer in the schools and was only superseded by the classical work, the *Fabrica* of Vesalius.

Barbarus, Hermolaus.
Castigationes Plinianae.
[Venice, unknown press for Daniel Barbarus, about 1493-94.]
One of the outstanding figures in the humanistic revolution of the XVth century. His castigations of Pliny the Elder's errors are an early example of the fully awakened critical spirit fighting against the tyranny of the ancients.

Gianmatteo Ferrari da Gradi.
Expositiones super 22 fen tertii canonis Avicennae.
Milan. Jacobus de Sancto Nazario de la Ripa. 1494, Nov. 17.
Gianmatteo da Gradi, about whom Albutt wrote so delightfully, in his *Greek Medicine and other Essays* was the master-mind of the old medieval school of Pavia. Though full of Arabic learning, fond of the hair-splitting methods of the Scholastics, he none the less recorded many shrewd clinical observations.

Argellata, Petrus de.
Chirurgia.
Venice, Unknown press. 1492, Apr. 28.
This is a second edition of Argellata's famous surgical work. The first appeared in Venice in 1480.

Institoris, Henricus and Sprenger, Jacobus.
Malleus maleficarum.
Nuremberg, Anton Koberger, 1494.
Institoris, alias Heinrich Kraemer, was a Dominican and a judge in witches' trials at the end of the XVth century in Germany. The fact

that this book is one of about ten XVth century editions shows the high esteem in which the author's skill in uncovering sorcery was held in those times.

Albertus Magnus.

Liber Physicorum.

Venice, Joh. de Forli & Gregorius fratres, 1494, ult. Jan.

At the end of the Middle Ages (XIIIth century) came Albertus Magnus and Roger Bacon, and with them began the new scientific revolution. They broke with traditional science, the former as systematic observer of nature, the latter as pioneer in experimental science. The *Physica* is a notable example of Albertus' work. But although a revolutionary, Albertus was also an erudite churchman. The *Summa* of his pupil Thomas Aquinas is still considered by some to show the deepest intellectual penetration into the secrets of nature.

Ketham, Johannes.

Fasciculus medicinae.

Venice, Johannes and Gregorius de Gregoriis, 15 Oct. 1495.

This "bundle" of medical tracts is one of the most famous of incunabula because of its beautiful woodcuts, drawn by a master of the School of Giov. Bellini. Ketham, a German physician of whom little is known, compiled the *Fasciculus*.

December, Petrus Candidus.

De genitura hominis.

[Rome, Stephan Plannck, 1495.]

As a physician and member of a family renowned for their humanistic learning, Pietro Candido wrote various treatises on medical subjects in Aristotelian vein. They are more remarkable for their Ciceronian Latin than for their contents.

Avenzoar (Ibn Zuhr) of Seville.

Libri III Theisir, i.e., interpretatio et rectificatio medicationis et regiminis. Antidotarium. Emendatus per. Hier. Surianus.

Averroes (Ibn Rushd) of Cordova.

Libri VII Colliget, i.e., de medicina. Emendat. per Hier. Surianus.

Venice, Bonetus Locatellus for Octavianus Scotus. 1496, ult. May.

Avenzoar (XIIth century) and Averroes are the two most famous Arab transmitters of classical learning. These two works, the *Theisir* and the *Colliget* (corruptions of the Arabic titles), were the basis for academic lectures on medicine during several centuries.

Petrus Hispanus (Pope John XXI).

Summulae logicae cum Versoris interposita expositione.

Venice, Bonetus Locatellus for Octavianus Scotus. 1496, July 12.

This is a work on logic by an author to whom is also attributed a *Thesaurus Pauperum*, or medical "treasure" book for the poor, a work

that had immense vogue from the XIIIth century on. It is not definitely decided whether the author of these two works is one and the same person nor whether it was he who later became Pope John XXI.

Cermisonus, Antonius.

Consilia with other medical tracts.

Venice, probably Bonetus Locatellus for Octavianus Scotus about 1496.

Cermisonus (died 1441) was professor at Padua and teacher of Giovanni Michele Savonorola. His *Consilia* which deals more with remedies than with the nature of diseases, is simply a mass of undigested facts.

Savonarola, Iohannes Michael.

Canonica de febribus.

Venice, Christoforus de Pensis. 1496, Oct. 16.

Iohannes Michael Savonarola (1390-1462), grandfather of the famous Florentine reformer, worked mostly in Ferrara. His clinical work marks a distinct departure from traditional scholastic teaching. He wrote various treatises which enjoyed great vogue.

Serapion, Yuhanna ibn ("Junior").

Breviarium medicinae. De simplici medicina. Dictus circa instans, practica Platearii.

[Venice]. Bonetus Locatellus for Octavianus Scotus 1497, Dec. 16.

Serapion was a highly esteemed Arabic author (of the XIth century) on pharmaceutical therapy. Little is known of his life.

Maimonides (Rabi Moyses = Musa ibn Maymun).

Aphorismi medici and other opuscules.

[Venice]. Bonetus Locatellus for Octavianus Scotus. 1497, Oct. 7.

Famous Jewish scholar of Cordova in the XIIIth century, who later became court physician to Saladin in Egypt. This book, containing his medical aphorisms together with other medical works, is a pirated reprint of a former edition by another printer, whose name was carelessly reproduced with the stolen text.

Leonicenus, Nicolaus.

Libellus de epidemia quam vulgo morbum Gallicum vocant.

Venice, Aldus Manutius, 1497.

Leoniceno was one of the most famous medical teachers of the Renaissance, in Padua and Ferrara. These three pamphlets deal with the most vital medical problem of the times, the outbreak of syphilis a few years previous.

Leonicenus, Nicolaus.

Libellus de epidemia quam vulgo morbum Gallicum vocant.

Milan, Gulielmus Signerre for Johannes de Legnano, 1497.

Leonicenus, Nicolaus.

Libellus de epidemia quam vulgo morbum Gallicum vocant.

[Leipzig, Wolfgang Stöckel, 1500.]

Maiolus, Laurentius.

De gradibus medicinarium.

Venice, [Aldus Manutius], 1497.

Maiolus lived in the XVth century in Genoa and wrote pharmaceutical works of which this, on the degrees and strength of medicines, is the principal. A good deal of speculation entered into this pharmaceutical work.

Argellata, Petrus de.

Cirurgia.

Venice, Bonetus Locatellus, 1497.

Pietro, one of the early pioneers of Italian surgery, taught in Bologna, where he died in 1423. His surgery in VI books is important, although he lays more stress on medicaments than on operations. He was the first to perform the incision of the linea alba in post-mortem Caesarian operations.

Arnoldus de Villanova.

Brevarium practicae medicinae.

Venice. Otinus de Luna. 1497. Oct. 21.

Arn. de Villanova, a Catalonian versed in Arabic learning, was a commanding figure in medicine during the XIIIth century. He taught in the Universities of Montpellier, Naples, and Paris. His brevarium contains many shrewd clinical observations besides traditional Arabic learning.

Brunschwig, Hieronymus.

Dis ist das buch der Cirurgia.

Augsburg, H. Schönsperger, 1497.

Bound with his

Liber pestilentialis.

Augsburg, H. Grüninger, 1500.

Brunschwig was a surgeon of Strassburg in the XVth and XVIth centuries. His *Chirurgia* was the first printed treatise on surgery and was abundantly illustrated. This is a piratical Augsburg reprint of the first edition which appeared in Strassburg in the same year. His plague tractate is derived from the earlier printed work by Steinhöwl.

Brandt, Sebastianus.

Stultifera navis.

Basel, J. Bergman, 1498.

The "fools' ship", written by Brandt in Latin and German verses, reflects as does Erasmus' *Praise of Folly*, the awakening critical spirit of the Renaissance, the revolt against merely traditional information. The woodcuts have been attributed to the young Dürer who was in Basel with Brandt.

Gordon, Bernardus de
Lilium medicinae.
Venice, Bonetus Locatellus, 1498.

Bernard of Gordon, probably a Scot, taught medicine in the University of Montpellier from 1285 on. In the preface to this popular medical treatise he indicates divisions by saying: "In this Lily there are many flowers and in each flower there are seven gold-like grains".

Guy de Chauliac, Lanfranc and others.
Collectio chirurgia Veneta.
Venice, Bonetus Locatellus, 1498.

Guy de Chauliac, the great medical teacher at Montpellier in the XIIIth century, laid, with some slightly earlier Italian and contemporary French colleagues, the foundation of French scientific surgery.

Albertus Magnus.
De virtutibus herbarum.
Cologne, printer unknown, ? 1499.

This pamphlet on the virtues of herbs and stones was erroneously attributed to Albertus Magnus, the great German philosopher and pioneer of science in the Middle Ages.

Astronomici veteres.
Venice, Aldus Manutius, 1499.

This finely printed collection of classical astronomy (mainly Alexandrian) came from the famous Venetian press of Aldus Manutius. By this and similar works he contributed powerfully to the revival of scientific studies. In this book there is a commentary of Sir Thomas Linacre written to Proclus Diadochus.

Boethius.
De consolatione philosophiae.
Lyons, Johannes de Vingle, 1499.

Boethius (about 480-524), Roman patrician and last of the great philosophers, was chancellor to the Gothic King Theoderic the Great. He wrote this consolation of philosophy while imprisoned at Pavia. He exerted great influence on the thought of the Middle Ages, through his translations and interpretations of classical works, among which are several scientific ones.

Isidorus Hispalensis.
Etymologiarum libri XX.
Paris, Thielman Kerver, 1499.

Isidorus Hispaliensis was bishop of Seville (about 570-636). He had immense learning and wrote many works, promoting the renewed interest in sciences generally and medicine especially. The present book, which appeared in countless editions, was a great authority throughout

the Middle Ages. His etymological interpretations are often forced and ridiculous, but the gathering of such a stock of knowledge insured the value of the book.

Brunschwig, Hieronymus.
De arte distillandi.
Strassburg, J. Grueninger, 1500.

Brunschwig was surgeon in Strassburg in the XVth century. His books on surgery and on the art of distilling medicines from herbs and other substances had an immense vogue. Grueninger who printed the present work is famed for his many earlier medical works, illustrated by woodcuts.

Bartholomaeus Anglicus.
Le proprietaire des choses, translated by Jean Corbichon.
[Lyons, probably Claude Nourry, 1500.]

Barthol. Anglicus, an English Franciscan monk, wrote this work on the properties of 'things' in the XIIIth century. It was probably the most popular compilation of the contemporary knowledge about natural objects. Nearly forty separate XVth century editions are known and there are translations into French, English, Dutch and Spanish. It contains chapters on anatomy, physiology and pathology. The English translation by Trevisa is most interesting. This very rare French edition is notable for the illustrations taken from manuscripts of which a good number are extant.

Bagellardus, Paulus, a Flumine.
De infantium asgritudinibus et remediis.
[Probably Lyons, Jean Dupré, XVth century.]

Bagellardo of Fiume, the author of this first pediatric primer, was professor at Padua in 1472. The chapters on the care of infants are particularly interesting.

School of Salerno.
Regimen sanitatis, with commentary of Arnoldus de Villanova.
Paris, Michel le Noir. 1497.

The great fame of this *Regimen Sanitatis* dates from much later times than is usually assumed. At the height of fame of the Salernitan School (XIth century), before Frederick II created the first medical code of laws, other "Rules of Health", *e.g.*, the famous "Aristotle letter", were in vogue. Arnoldus de Villanova, who was in Salerno in the XIIIth century, probably popularized this *Regimen* in verse, which lasted through into our times.

School of Salerno.
Regimen sanitatis.
Venice, B. de Vitalibus, not after 1500.

Hippocrates.

Various tracts.

[Rome, S. Plannck, 1500.]

We show two little books of the same place and printer, with slight variations.

During the period of incunabula (1460-1500) very few works of the Father of Medicine were printed. Only tracts, many erroneously attributed to him to promote their sale, were issued. This is an interesting specimen edited by Andreas Brentius, the powerful and learned churchman who lived at the time of the humanistic Pope Sixtus IV.

ANNOUNCEMENTS

The Library is anxious to increase its collection of medical portraits. Fellows are requested to send to the Library their photographs and other medical portraits which they might like to present.

There are a good many gaps in our collection of medical thèses. We should be very pleased to receive gifts of such thèses from Fellows of the Academy and others, if they can spare them.

PROCEEDINGS OF ACADEMY MEETINGS

FEBRUARY

STATED MEETINGS

Thursday Evening, February 7, at 8:30 o'clock

Program presented in coöperation with the Section of Surgery

ORDER

I. EXECUTIVE SESSION
Action on amendment to the By-Laws introduced at Stated Meeting of January 3, 1929
Election of Fellows

II. PAPERS OF THE EVENING
a. Surgical treatment of gangrene and infection of the feet in diabetes, Leland S. McKittrick, Boston (by invitation)
Discussion, Henry J. Spencer, John Douglas
b. Spinal anesthesia in abdominal operations, Lincoln F. Sise, Boston (by invitation)
Discussion, George P. Pitkin (by invitation), Malcolm T. Munkittrick (by invitation)

Thursday Evening, February 21, at 8:30 o'clock

THE FIFTH HARVEY LECTURE

Constitutional variation and susceptibility to disease

WADE H. BROWN
Member of The Rockefeller Institute for Medical Research, New York, N. Y.

Peyton Rous, President
Harvey Society

Philip D. McMaster, Secretary
Harvey Society

This lecture takes the place of the second Stated Meeting of the Academy for February.

SECTION MEETINGS

SECTION OF DERMATOLOGY AND SYPHILIS

Tuesday Evening, February 5, at 7:45 o'clock

ORDER

I. DR. HOWARD FOX gave a talk illustrated with lantern slides on yaws as seen in Haiti.

II. PRESENTATION OF PATIENTS
a. Cases from the Skin and Cancer Hospital, Paul E. Bechet, A. Schuyler Clark, Binford Throne, Chas. M. Williams, David S. D. Jessup, Eugene F. Traub, Herman Goodman, Wm. Bayard Long, Hermann Feit
b. Miscellaneous Cases

III. Discussion

IV. Executive Session

Section of Otology

Friday Evening, February 8, at 8:30 o'clock

ORDER

I. Reading of the Minutes

II. Presentation of Instrument
Modification of the eustachian tube catheter, George M. McAuliffe

III. Papers of the Evening
a. The window operation for hematoma auris and perichondritis with effusion, Robert C. Howard
Discussion, George E. Davis
b. The modified radical mastoid operation for chronic otorrhea (Lantern slides), Hugh B. Blackwell
c. Newer developments in neuro-otology, Lewis Fisher, Philadelphia (by invitation)
Discussion, Randal Hoyt (by invitation)

IV. General Discussion

V. Executive Session

Section of Neurology and Psychiatry

Tuesday Evening, February 12, at 8:30 o'clock

ORDER

Program Presented by the Department of Neurology, Columbia University

I. Reading of the Minutes

II. Presentation of Cases
a. A case of oxycephaly, Samuel Atkin (by invitation)
b. A series of cases of brain tumors, Charles A. Elsberg

III. Papers of the Evening
a. Syphilitic hemorrhagic meningo-encephalitis illustrated by pathological material (lantern slides), Irving J. Sands
Discussion, Armando Ferraro (by invitation), Leon H. Cornwall
b. Tumors of the spinal cord. Problems in their diagnosis and localization. Procedures for their exposure and removal, Charles A. Elsberg

Section of Pediatrics

Thursday Evening, February 14, at 8:30 o'clock

ORDER

I. Papers of the Evening
a. Beriberi in late infancy, Sidney V. Haas
Discussion, Bela Shick

b. The natural history of rheumatic infection; evaluation of therapeutic measures, May G. Wilson
Discussion, Homer F. Swift
c. Appendicitis in children, Charles E. Farr
Discussion, Richard W. Bolling

Section of Orthopedic Surgery

Friday Evening, February 15, at 8:30 o'clock

ORDER

I. Reading of the Minutes

II. Presentation of Cases
a. Cases demonstrating injection treatment of varicose veins, J. S. Tunick (by invitation)

III. Papers of the Evening
a. Injection treatment of varicose veins and ulcers, J. S. Tunick (by invitation)
b. A quantitative tuberculin test for active tuberculosis, W. I. Galland (by invitation), J. E. Blair (by invitation)

Section of Ophthalmology

Monday Evening, February 18, at 8:30 o'clock

ORDER

I. Reading of the Minutes

II. Clinical Cases
a. Exophthalmus due to hematoma of frontal sinus, Lewis Webb Crigler
b. Report of a case of sympathetic ophthalmia occurring 48 years after injury, with microscopical sections, James W. Smith

III. Demonstration
A plan for graphically recording motor anomalies, James W. White

IV. Paper of the Evening (9 p.m.)
The value of tuberculin in ophthalmology, Clarence King, Cincinnati (by invitation)
Discussion by George S. Derby, Boston (by invitation), John E. Weeks, James G. Dwyer, Andrew A. Eggston, A. B. Ferguson (by invitation)

V. Executive Session

Section of Medicine

Tuesday Evening, February 19, at 8:30 o'clock

ORDER

I. Papers of the Evening
Symposium on Asphyxia

a. Carbon monoxide asphyxia and its treatment, Yandel Henderson, Yale Medical School (by invitation)

b. Modern methods of resuscitation in New York City emergencies, with motion pictures, Daniel J. Donovan, Chief Surgeon, N. Y. Police Dept.

A demonstration of the Prone Pressure Method will be given by the Police Emergency Squad.

Discussion by Alvan LeRoy Barach, Charles Norris, Harry M. Archer (by invitation)

Section of Genito-Urinary Surgery

Wednesday Evening, February 20, at 8:30 o'clock

ORDER

I. Reading of the Minutes

II. Presentation of Cases

a. 1. Solitary cyst of kidney—nephrectomy—lantern demonstration
2. Teratoma testis—orchidectomy—specimen, R. L. McKiernan (by invitation)

b. Ectopic kidney—nephrectomy—specimen, Clyde W. Collings

c. Exhibition of a new cysto-urography table, Frederick E. B. Foley, Saint Paul, Minn. (by invitation)

III. Paper of the Evening

Urologic and surgical care of nephrolithiasis, James N. Vander Veer, Albany (by invitation)

Section of Obstetrics and Gynecology

Tuesday Evening, February 26, at 8:30 o'clock

ORDER

I. Reading of the Minutes

II. Presentation of Cases

a. A case of endometriosis with polypoid invasion of the vagina (with lantern slides), Samuel H. Geist

Discussion by Robert Tilden Frank

b. Report of cases of pyometra past the climacteric, Hermann Grad

Discussion by Isidor Kross

III. Papers of the Evening

a. Vaginal operations in the treatment of uterine prolapse, cystocele and rectocele, with special reference to the interposition operation, Louis E. Phaneuf (by invitation)

Discussion by George Gray Ward, William P. Healy, Byron H. Goff

b. Obstetrical anesthesia, S. A. Cosgrove

Discussion by Paluel Joseph Flagg (by invitation), James Tayloe Gwathmey

Discussion of papers and cases is limited to Fellows of the Academy and specially invited guests.

SECTION OF LARYNGOLOGY AND RHINOLOGY

Wednesday Evening, February 27, at 8:00 o'clock

(Please note change of hour)

MT. SINAI HOSPITAL NIGHT

ORDER

I. READING OF THE MINUTES

II. PRESENTATION OF PATIENTS
- a. Lung abscess cured by bronchoscopy
- b. Lung abscess cured by bronchoscopy
- c. Lung abscess cured by bronchoscopy, M. S. Bender (by invitation)
- d. Osteoma of antrum
- e. Carcinoma of nasopharynx, L. Kleinfeld (by invitation)
- f. Lateral cervical fistula
- g. Orbital abscess following ethmoiditis; intranasal operation
- h. Lingual thyroid
- i. Carcinoma of eustachian tube; radium implantation
- j. Extrinsic carcinoma of larynx; radical operation
- k. Extrinsic carcinoma of larynx; radical operation, Rudolph Kramer

III. REPORT OF CASES
- a. Benign tumor of bronchus, Sidney Yankhauer
- b. Basal cell carcinoma of antrum with pulmonary metastasis L. Kleinfeld (by invitation)

IV. DEMONSTRATIONS
- a. Simplified investigation of the sphenoid sinus, Mervin C. Myerson
- b. Irrigation of the antrum in infants and children by means of the natural orifice cannula, H. G. Goldman (by invitation)

V. PAPERS OF THE EVENING
- a. Intracranial complications following sphenoid infections, Rudolph Kramer
- b. Pathological report of two cases of intracranial complications associated with infection in the sphenoid (lantern slides), J. G. Druss (by invitation)

NOTES

Catalogue of Third Annual
EXHIBITION OF WORKS IN THE PLASTIC AND GRAPHIC ARTS BY AMERICAN PHYSICIANS

Under the Auspices of the New York Physicians Art Club

Held at The New York Academy of Medicine, February 2nd to 15th, 1929.

Introduction

Linsly R. Williams

In the early winter of 1927, a very unusual exhibition was held at the new building of The New York Academy of Medicine and as far as is known, it was the first exhibition of the kind held in the United States.

The Physicians' Art Club prepared for exhibition a variety of artistic objects which included portraits, paintings, oils, and water colors, sculpture, book plates, engravings, etchings and a variety of other interesting objects of art, all of which were executed by medical men.

This exhibit raised a great deal of interest not only among the medical profession but amongst the general public.

A similar exhibit was held in 1928 and a third is to be opened on February first under the same auspices.

A deep interest has been taken in these exhibits by the Council and Fellows of the Academy because it demonstrates that many physicians have their avocations as well as their professional vocation. It is not unusual for trained physicians to become well known in the field of art and letters. The exquisite beauty of Hayden's etchings is well known to many art students, but how many know that he was a qualified physician. The writings of Rabelais, Smollet, Holmes and Mitchell have made these physicians far more celebrated than have their professional activities, although Holmes was celebrated as an anatomist and Mitchell as a neurologist.

Gerald Webb of Colorado Springs has recently pointed out that the training of physicians stimulates their imaginations and artistic sense far more than does the study of the other liberal professions. Whether this be true or not, the fact remains that at the present time there are physicians living in England and in this country and in many other parts of the civilized world whose devotion to the arts and letters is almost as keen as their interest in their own chosen field of endeavor.

This exhibit is but another demonstration of the medical interest in non-professional affairs and it is hoped that they may be continued and will stimulate the members of this broad profession to develop an avocation which is worthy of its best traditions.

List of Works

ALLEN, JOHN H. (Portland)
- 3 Still Life

AYER, JAMES C. (New York City)
- 4 Nude Study
- 5 Portrait of Miss Sims
- 6 Still Life

BANCEL, HENRY A. (New York City)
- 7 Morning Landscape
- 8 House with Five Chimneys
- 9 Nantucket Fishing Boat

BARRINGER, B. S. (New York City)
- 10 Golden Fall
- 11 Panel
- 12 Old Apple Tree

BAUCH, S. (STAN.) (New York City)
- 13 Sails
- 14 Lake Tahoe
- 15 Sea Gate

BERKOWITZ, JOSEPH J. (New York City)
- 16 The Doctor, Model 1928

BURKE, EDGAR (Jersey City)
- 17 Canvasbacks
- 18 Ring-necked Pheasants

CHAMPLIN, H. H. (New York City)
- 19 The Sigh
- 20 "Thrown on Her Hundred Isles" (Byron)

COHN, SALO (New York City)
- 21 Summertime
- 22 In the Catskills

CUNNINGHAM, THOM. H. (Glens Falls)
- 23 End of the Day
- 24 Scrub Pine (Etching)

DICKINSON, ROBERT L. (New York City)
- 25 Views from the Windows of The Academy of Medicine
- 26 Bookplates, Pasters and Christmas Cards
- 27 Sunset Over Moab and the Wilderness from the Mount of Olives

DOLGOPOL, VERA B. (New York City)
- 28 Etude
- 29 Etude

ELLIOTT, GEORGE R. (New York City)
- 30 Box Made of St. Domingo Mahogany which came to New York in George Washington's Time

FISCHER, HERMANN (New York City)
- 31 October Day
- 32 Waxenstein (Etching)
- 33 Il Castello, Capri (Etching)
- 34 Mountain Lake (Etching)

FREEMAN, ALPHEUS (New York City)
- 35 The Boat Yard (Loaned by Dr. George P. Willis)
- 36 The Omar Khayyam (Loaned by Dr. George P. Willis)
- 37 Wincoma

FRIDENBERG, PERCY (New York City)
- 38 Thames Barges
- 39 Cornwall

GRANET, H. (New York City)
- 40 Lovers (Placque)

HARTSCHORN, W. MORGAN (New York City)
- 41 The Birches
- 42 Landscape
- 43 Farm, Cornwall, Conn.

HIRSCH, I. SETH (New York City)
- 44 Portrait of Mrs. H.
- 45 Moses (Sculpture)

HOLDEN, WARD A. (New York City)
- 46 Sunset
- 47 Sunset
- 48 Morning

HUNT, LEIGH (New York City)
- 49 Etaples
- 50 A Street Scene
- 51 The River Bank

52 Low Tide
53 Night—Picardy
54 In the Mist
55 In a Canal
56 Mattituck Creek

JAEGER, CHARLES H. (New York City)
57 The Swans of Bruges
58 Phantom Ship
59 Fallen Monarch

KAHN, MORRIS H. (New York City)
60 Portrait Sculpture
61 Girl's Head
62 Entwined

KEYES, STANLEY J. (New York Ctiy)
63 "Home with Mainsails Backed and Bows a Sea of Foam"
64 Morning Mist
65 "Out Beyond the Sunset Could I But Find the Way."—
John Masefield

KILMER, T. W. (New York City)
66 Portrait of Pirie MacDonald
67 Richard Abbott as Abraham
68 Portrait of Mr. R.

KOCH, ROBERT E. (New York City)
69 Chess Board—An Example of Handmade Marquetry, Containing
8260 Pieces of Wood

LARRABEE, RALPH C. (Boston)
70 September Hay
71 Scrub Pines

LAVANDERA, MIGUEL (New York City)
72 A Panama Hat

LEVINE, GEORGE G. (New York City)
73 Santa Maria

LILIENTHAL, HOWARD (New York City)
74 Charge

LYNN, ETHEL (Mrs. Harlo Lynn) (San Francisco)
75 A California Giant
76 Desert Sunset

MALONEY, DANIEL J. (Waterbury, Conn.)
77 Southington Mountain

MARTINETTI, C. D. (Orange, N. J.)
78 Sand Dunes
79 Evening

McCREADY, JAMES A. (New York City)
80 Sylvan Stream in Afterglow

81 Sycamores in Sunlight
82 Beethoven

MINSKY, HENRY (New York City)
83 Fundus
84 Fundus

MOSSMAN, JAMES (New York City)
85 Portrait (Plaster)
86 Thunder on the Right
87 October Hills
88 Burton's Farm
89 Red Boat-House
90 Balsams
90A Approaching Rain

NILSEN, ARTHUR (New York City)
91 Drifting In
92 Portrait—"Alexander Popini"
93 Portrait—"Sir St. Clair Thomson"

OASTLER, FRANK R. (New York City)
94 Dall Ram
95 California Redwoods
96 Gulf of Alaska

PATTERSON, HENRY S. (New York City)
97 Dawn, Eastport
98 Autumn in Ramapo Hills
99 Boblainey Wood, Invernes

PIERCE, GEORGE HOWARD (New York City)
100 Beyond the Horizon
101 The Approaching Storm

SCHROEDER, LOUIS C. (New York City)
102 Portrait Bust of Boy

SMITH, ALONZO DeG. (New York City)
103 North African
104 Old Man Grey

STRONG, A. McI. (New York City)
105 South Sea Beach

TARTAKOW, I. J. (New York City)
106 Bookplate: "The Talmudist"
107 Charcoal Study of an Infant

WIENER, RICHARD E. (Florence, Italy)
108 Still Life
109 Asters
110 Peonies (Loaned by Mr. and Mrs. S. M. Lehman)
111 Fruit and Flowers

WIGHTMAN, ORRIN S. (New York City)
112 Six Portraits

WOGLOM, WILLIAM H. (New York City)
113 William
114 The River
WOLFE, BERAN W. (New York City)
115 Pensive Head
116 Mask
117 Head of a Young Girl
118 The Psychiatrist (Drypoint)
119 The Danube (Etching and aquatint)
WOLF, MAX (New York City)
120 Act (Oil Sketch)
121 Etchings
122 Etchings
McKENZIE, R. TAIT (Philadelphia)
123 Tortoise Doorknocker
MOSHER, HARRIS P. (Boston)
124 The Mooring
125 Bunker Hill
126 The Floyd Ireson House, Marblehead

NOTICE

Beginning in February the meetings of the Society for Experimental Biology and Medicine will be held under the auspices of The New York Academy of Medicine.

DEATHS OF FELLOWS OF THE ACADEMY

Victor Hugo Jackson, M.D., 57 West 57th Street, New York City; graduated in medicine from the University of Michigan, Ann Arbor, Michigan, in 1878; elected a Fellow of the Academy April 6, 1893; died, January 26, 1929. Dr. Jackson was a Fellow of the American Medical Association and a member of the National Tuberculosis Association.

Walter Barry Jennings, M.D., Connecticut State Hospital, Middletown, Connecticut; graduated in medicine from New York University, New York City, in 1898; elected a Fellow of the Academy February 1, 1906; died, January 21, 1929. Dr. Jennings was a Fellow of the American Medical Association, a member of the American Psychiatric Association and of the Connecticut State Medical Society.

JOSEPH RANKIN LOSEE, M.D., 114 East 54th Street, New York City; graduated in medicine from the Long Island College Hospital Medical College, Brooklyn, New York, in 1907; elected a Fellow of the Academy February 1, 1917; died, February 4, 1929. Dr. Losee was a Fellow of the American Medical Association, Pathologist and Medical Executive Officer of the Polyclinic Medical School and Hospital, and Pathologist to the Lying-in Hospital, New York.

FREDERICK SAMUEL LOVELL, M.D., 31 West 74th Street, New York City; graduated in medicine from New York University, New York City, in 1886; elected a Fellow of the Academy November 2, 1911; died February 7, 1929. Dr. Lovell was a Fellow of the American Medical Association, Rhinologist and Laryngologist to the Polyclinic Medical School and Hospital, Consulting Surgeon to the Bronx Eye and Ear Hospital and Consulting Nose and Throat Surgeon to St. Catherine's Hospital.